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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—11TH YEAR. SYDNEY: SATURDAY, DECEMBER 13, 1924. No. 24.

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KIDNEY FUNCTION IN HEALTH AND DISEASE.[1]

By J. V. DUHIG, M.B. (Sydney),
Brisbane.

RECENT research in human biology has been for the most part along chemical rather than anatomical and histological lines and has given us a more detailed view of normal function and a better appreciation of the mechanism of morbid processes of which formerly we apprehended the significance, but which owing to limited methods of investigation, eluded exact definition. Especially in nephritis we were dominated too much by what we found to exist after death and diagnosis and prognosis were founded on a purely conjectural anatomical classification. To condemn a man to invalidism because his urine contains albumin is not now scientific and with a view of helping in a small way to put the present position clearly before you, I give this short paper as a commentary on the specimens of morbid kidneys I am showing this evening.

That albuminuria is not in itself evidence of grave disturbance of kidney function, is well known, but what then constitutes nephritis, the disease formerly considered synonymous with that sign by able men and still so considered by the insurance companies, is perhaps not quite so clear. At the beginning I want you to dismiss from your minds the old anatomical classification of nephritis, since it has ceased to have any but secondary importance in the investigation of the disease. It seems unfair to diagnose your patient's illness by what you are going to find in him on the *post mortem* table—by end results. Even if your patient have albumin in his urine, some casts, no chlorides and only a little urea, he may still outlive you. In the light of our present knowledge it is hazardous to try to give an exact prognosis by rule of thumb. Each case of nephritis is a complete chemical study in itself. What we have to decide is how far the destructive process has gone and how much further it is likely to go in each particular instance, basing our opinion on the actual findings in that case and none other. I have found *post mortem* fibrous kidneys in men whose history of real illness could be reckoned in months, but whose *post mortem* signs should, according to our former criteria, have taken years to evolve. I mean that an arterio-sclerotic kidney, so-called, may arrive at the final goal of its mission in almost as short a time as an acute glomerular or tubular nephritis may take to destroy life. By that I mean that the toxic agent of nephritis may act swiftly or shortly and by different routes or selectively as it were on different structures. That there

[1] Read at a meeting of the Queensland Branch of the British Medical Association on October 3, 1924.

may be a broad general classification of selective action and so of prognosis I do not deny, but I have seen so many cases that on established principles were very perplexing and I may say disappointing that I think it is dangerous to base treatment and prognosis on purely cursory clinical data alone. I think it is necessary to establish first the extent of the lesion, then to estimate its progress and after repeated trial only to give a prognosis. Now it is possible to do this with a certain amount of accuracy, though what is the ætiologocal agent which either makes the glomerulus permeable to plasma proteins or the uriniferous tubules impermeable, I cannot say. But what we can say is how the normal kidney behaves and with a certain degree of completeness how the body reacts chemically when the toxin is at work.

The kidney is a complex of many different structures, glomeruli, uriniferous tubules of varying structure, nerves and blood vessels, supported by a framework of connective tissue; all these mutually helpful combine to throw out chemical compounds which are either useless in themselves, being poisonous end products or are useful but present in excess. How the urine is exactly compounded or excreted or perhaps secreted, is still a complete mystery. All we know is that its composition varies within wide limits and we can estimate the general effect of these variations. The most reasonable theory of urinary secretion is by consent admitted to be that of Cushing[1] who regards the glomerulus as a simple filter dependent naturally on the two factors of blood pressure and blood flow to produce sufficient "head" to cause filtration from the glomerular capillaries into the capsule. The glomerulus normally is permeable only to soluble crystalloids, that is to say that the urine filtered into the capsule is a solution of the soluble salts of the blood minus the blood solids and proteins of the plasma. It is then necessary to regard the glomerular urine as containing in solution the soluble substances of the plasma in the same concentration as in the blood. We know, however, that the urine contains these substances in a concentration many times that of the blood. This urinary concentration is apparently carried out by reabsorption of water from the tubules along with soluble "threshold" substances as chloride and sugar, in normal persons. This theory is not altogether satisfactory, as Maclean has shown, since reabsorption of water should leave the concentration of soluble substances constant for all and leave the concentration of "threshold" substances in the urine at a level in constant relation to the "threshold" level of excretion in the blood. This does not always occur, as can be seen in diabetes and diseases associated with chloride retention. But the theory does give some sort of idea of what occurs in the kidney, especially in nephritis, particularly when diuresis is low and casts are numerous. It is easy to imagine casts as desiccated albumin remaining in the tubules after re-absorption of water and in that respect the theory is acceptable.

The function then of the kidney is to excrete waste products from the body, either those of which it has no further need, end-products or substances in excess of the amount required to sustain life—"threshold" substances. It is very important to appreciate the difference. "Non-threshold" substances will be excreted in the urine always, even when they are present in the blood in low concentration as urea, uric acid, creatinin, whereas the tubules will restore to the blood those substances which are essential to life, as glucose, chlorides and water. These substances are normally not excreted in the urine until they have reached a certain concentration in the blood. Sodium chloride does not appear in the urine until it has reached a concentration in the blood of 0.562%. Any chloride in the blood in excess of that will be excreted under normal conditions and, conversely, if the chloride level in the blood tends to fall below this proportion, the kidneys will respond by excreting water until the normal "threshold" level is restored. This has just as important a bearing on treatment in nephritis as the glucose "threshold" has in diabetes. Besides this power to excrete waste and superfluous normal substances, the kidney plays a most important part in maintaining the delicate acid-base equilibrium of the blood represented by the ratio $\frac{H_2CO_3}{NaHCO_3}$, which is normally constant, both acid and base being constant. Normally the hydrogen-ion concentration of the blood is constant, close to $P_H = 7.4$, kept so by the protective action of the bicarbonates and phosphates and to some extent also by the solids and proteins of the blood. Even the slightest variation of this ratio is the result of grave disturbance, so that we gather that the mechanism for the maintenance of equilibrium is extremely delicate and adaptable. This mechanism is bound up with the excretion of acid waste or acid excess through the bowels, through the lungs as carbon dioxide and through the kidneys especially. Failure to excrete these acids results, if uncompensated by increase of alkali from the alkali reserve or otherwise, in what we term "uncompensated acidosis," which simply means that the P_H value of the blood is decreased, which may occur either by increase of carbonic acid without corresponding increase of sodium bicarbonate or by decrease of the alkaline reserve without a corresponding decrease of carbonic acid, an "uncompensated alkali deficit" being the term we apply to the latter condition, such as occurs in the terminal stages of nephritic acidosis.

The term acidosis, originally applied to diabetic ketosis, is now used in a broader sense, but it is not quite exact. What we really mean to express by the term is that the hydrogen-ion content of the blood is increased, that is the acid-base balance is disturbed so that the $\frac{H_2CO_3}{NaHCO_3}$ ratio is increased, not that in any sense the blood becomes grossly acid. Acidosis may result from either an abnormal formation of acid substances as in diabetes or from decreased elimination of acid substances normally present in the blood. When this occurs the body must draw on its alkali reserve to compensate, but

the kidneys in these conditions are able to secrete an acid urine from nearly neutral blood by breaking down dibasic phosphate (Na_2HPO_4) into sodium hydrate (NaOH) and acid sodium phosphate (NaH_2PO_4), the alkali being retained and resorbed from the tubules and the acid phosphate excreted. It is thus that the kidneys help to maintain the normal balance and it is inability to do that in the terminal stages of nephritis which precipitates acidosis, nephritic acidosis being in effect the result of phosphate retention.[2] Normally the kidneys secrete an acid urine and it has been found that the acidity lies exactly between that of the dibasic and the acid sodium phosphate, since the kidney concentrates many times the acid phosphate and returns the alkali to the tissues. As a further contribution to the maintenance of an alkali reserve it excretes ammonia in preference to more stable and more necessary alkali radicles. This is well shown by the high ammonia excretion in diabetic acidosis to compensate for the alkali deficit produced by neutralization of acid ketones.

Finally the kidneys constantly maintain the chloride of the blood at a constant level. The slightest variation in chloride intake is at once compensated by excretion of (*a*) chlorides if the level tends to rise over the "threshold" or (*b*) water if the chloride level tends to fall; it is thus kept at a concentration normally of about 0.56%. Now this function is disturbed in nephritis and the use of chlorides in treatment is a matter of some moment and delicacy.

The function then of the kidney is to excrete such waste products as urea, uric acid and purin bodies, to excrete normally formed acids as acid phosphate, to save stable alkali for the tissues by excretion of ammonia, to restore to the body substances required by it in certain concentration as sugar and chlorides and lastly to hold back necessary nitrogenous substances, the blood proteins.

In disease of the kidney one or all of these functions may break down and I propose to discuss shortly the structural alterations resulting and the chemical changes that ensue and how we may estimate them directly or indirectly. In acute toxic nephritis all the elements concerned in secretion are probably involved. We may then have an acute tubular nephritis, an exudative glomerulo-nephritis or intra-capillary glomerulitis. The blood vessels also become involved in the change as a capillary lesion near or within the tufts or later as a degenerative and obliterative change in the larger arteries. Clinically it is impossible to say what particular structures are most involved. The process is bilateral and probably complete in both organs since the process starts from a soluble toxin. The result is such as we might expect. The glomerular filter ceases to function and at the same time is so damaged that it permits protein to pass through. Water is resorbed in excess of the normal amount and as a result urea, uric acid and salt are retained. Though urea may be concentrated to the normal amount, its output is absolutely diminished, so that it accumulates in the blood. This urea retention is constant in acute nephritis. Chlorides as a rule are retained and seem to bear a distinct relationship to the œdema which is usually present. Resorption of water from the tubules leads to desiccation of the protein and casts come down in the urine with blood from the damaged capsule or tuft; so that we have an interesting chemical picture in the early stages—scanty blood-stained urine with much protein, blood, casts, urea in probably normal concentration and no chlorides. The oliguria persists and later large quantities of urine are passed containing more urea and more chloride than normal. This may occur even though the lesion persists, probably for the reason that urea is an excellent diuretic and when highly concentrated in the blood produces diuresis. The chlorides later are excreted usually at first in excess until the normal "threshold" level is reached. Chloride retention bears, as I say, a distinct relation to fluid retention and the hydræmia which is a constant feature of the acute disease. While œdema and hydræmia persist naturally, chlorides will be withheld as we withhold nitrogen from the diet, not so much, I suppose, to reduce the actual volume of the protein excreted as to reduce its poisonous effect on the glomeruli. There are only two methods of estimating the prognosis. The first is to test the blood urea concentration. As it falls on a fixed dietary, the prognosis improves. It is useless, however, if the nitrogen intake is varied from test to test. The second is to employ the phenol-red test. In acute cases or chronic cases of bady damaged kidney there is distinct lowering of concentration in two hours.

As the hydræmia decreases, so naturally the blood pressure falls and the "head" of blood on the glomerular filter decreases with all round improvement. The acute condition may clear up absolutely as we know happens after, say, a metastatic streptococcal infection or it may clear up and relapse and become chronic.

It is easy to imagine what structural changes may occur after an acute nephritis, some of the secreting tissue being completely obliterated by the scar tissue laid down in the process of repair or all of it may ultimately become involved. This is the type of lesion seen most commonly in young people, which seems so hopeless to treat with success.

Various types of anatomical change are seen from a small red granular kidney to a large pale type, depending on the site of origin of the process. In general it may be said that any combination of lesions may exist between the small red or white granular kidney and the large, pale type and it is practically impossible under present conditions to decide precisely the nature of the lesion and how it stands. There are the two extremes, a progressive toxic chronic nephritis originating from an acute attack and usually rapidly fatal and the insidious chronic type usually called "interstitial" (not very correctly, I think), which is not so rapidly progressive. Between these extremes any combination may occur and it benefits nothing nor is it at present scientific to attempt to anticipate the microscopical

findings after death. But if histological classification is uncertain and unsound, it is especially in these chronic cases that we learn so much of the vital chemistry of urine secretion.

In the severer form found in young subjects the clinical picture is interesting in view of the chemical findings. These patients never feel well, are occasionally slightly dropsical, have no appetite, constantly suffer from morning headache, look pale and puffy and go down hill rapidly. We know that their kidney function is disturbed and that disturbance is reflected in many ways. Obviously from what we know of normal function the body is constantly striving to maintain the normal acid-base balance and what the kidney tries to do is also rather curiously reflected in the symptoms. We know these patients pass more urine in the nocturnal twelve hours than during the rest of the day. Why is that? I think the explanation must be that, as Calvert, Mayrs and Milroy[3] have shown, there is normally a physiological acidosis during sleep, the PH value of the blood moves down to the acid side. That occurs normally and in the nephritic whose glomerular filter is destroyed, probably there is in addition to that some acid phosphate retention. Under this stimulus and the increased "head" of acid required to produce secretion, urine is formed to get rid of that excess, which is, you will remember, higher than in the day time because of that normal physiological fact. Diuresis occurs. Then another interesting thing happens. The same authors have shown that the effect of diuresis is to decrease the monobasic-dibasic phosphate ratio, that is the urine tends to become alkaline by increase of ammonia and other alkalis. And the result is that the alkali reserve is further lowered and the PH value of the blood moves down and the patient gets his morning headache and feels wretched; he has an acidosis. Whether it would be a good thing, therefore, to prescribe an evening dose of non-diuretic alkali, I do not know, but I should like to see it tried. The same authors show that it has a considerable effect on the phosphate ratio. In these cases again chloride is retained sometimes and œdema results, leading to hydræmia, with increased blood pressure and anæmia, a lowered hæmoglobin value and colour index. These patients too retain urea, but it is fallacious to rest content with a normal urea concentration reading from the urine, since far from indicating that the urea excretion in the urine is an index of blood urea fall, exactly the opposite is the case for the very good reason that kidney filtration being faulty, it requires a much larger "head" of urea, sometimes ten times normal, in the blood to force a normal concentration through the kidneys. It is in this connexion that Maclean's modification of Ambard's formula is so helpful. Dye excretion is at a minimum and by the "water test" fixation of specific gravity occurs. Normally according to fluid intake the specific gravity can be varied almost at will. These patients never do well. Acid phosphate retention persists, until the alkaline reserve is so diminished that the carbon dioxide combining power is lost and the patient dies comatose. This conditoin is of kaleidoscopic chemical interest and so many variations of symptoms and reactions occur that each case should be treated on its merits. We should from time to time measure the day and night urine, test for specific gravity fixation, the amount of albumin, the urea in the blood and its concentration in the urine, the chloride excretion and the influence of dietary nitrogen on all these.

Lately much attention has been directed to the estimation of the uric acid blood content in

TABLE I.

Case.	Age (Years).	Sex.	Date.	Uric Acid (Milligrammes per 100 Cubic Centimetres)	Urea (Milligrammes per 100 Cubic Centimetres)	Remarks.
1	55	male	18/3/16	6.1	16	Chronic diffuse nephritis.
2	25	female	15/3/16	9.6	19	Chronic diffuse nephritis.
3	56	male	7/6/15	7.1	16	Chronic interstitial nephritis.
			21/7/15	6.6	24	
			28/9/15	6.3	18	
4	65	male	11/8/15	9.5	25	Chronic interstitial nephritis.
			25/9/15	8.0	37	
			14/1/16	5.0	37	
5	11	male	27/6/16	8.0	36	Chronic diffuse nephritis.
			21/7/16	6.3	53	
6	49	male	15/1/16	9.5	44	Acute nephritis, acidosis, recovery.
			28/1/16	2.5	19	
7	8	male	18/12/17	11.4	106	Acute nephritis, anuria, recovery.
			21/12/17	11.2	93	
			27/12/17	5.0	21	
8	21	female	4/12/19	9.3	48	Chronic interstitial nephritis, died.
9	37	female	19/3/20	7.7	111	Chronic interstitial nephritis, died.
10	62	male	4/4/16	3.4	28	Chronic parenchymatous nephritis.
11	27	female	17/4/20	11.0	13	Eclampsia, died.
12	20	female	20/12/20	6.9	6	Eclampsia, died.

(Taken from Myers's "Practical Chemical Analysis of Blood," Second Edition, page 53.) It will be noticed that the uric acid and urea run roughly parallel.

TABLE II.

Patient.				Urine.										
Initials.	Age, Years.	Sex.	Date.	Period.	Volume (Cubic Centimetres)	Specific Gravity.	Phosphate Ratio.[1]	Albumin.	Casts.	Urea.	Chlorides (Grammes per Litre).	"Water Test."	Blood Uric Acid (Milligrammes per 100 Cubic Centimetres)	Phenol Red Test.
F.T.	20	male	October 13	night	340	1020	—	+	—	—	—	—	—	—
						1026	—	—	—	—	—	—	—	—
			October 14	day	653	1012	5 : 5	+	granular.	—	—	10 20	—	—
						1011	—	—	hyaline	—	—	00	—	—
				night	866	1012	4.8 : 5.2	+	granular,	2.28%	—	00	—	{10 / 8.5}[2]
						1011	—	—	hyaline	—	—	10	—	—
			October 15	day	810	1015	1.1 : 8.9[3]	—	—	—	—	—	—	—
				night	840	1020	5.2 : 4.8	+	hyaline,	—	—	—	—	—
			October 16	day	924	1015	1 : 9[3]	—	cylindrical	—	—	—	—	—
				night	280	1025	—	—	—	—	—	—	—	—
			October 17	day	—	—	—	—	—	—	—	—	—	—
				night	476	1020	7.5 : 2.5	+	granular,	—	15.4[4]	—	—	—
			October 18	day	—	1020	4 : 6	+	hyaline	—	9.5[5]	—	3.6	—
									granular,					
									hyaline					
M.M.	22	male	October 13	night	653	—	5.4 : 4.6	trace	none	—	—	—	—	—
			October 14	day[6]	577	—	—	—	—	—	—	—	—	—
				night	—	1020	6.6 : 3.4	—	—	—	—	25 10	—	—
												05 20		
			October 15	day	—	—	2.6 : 7.4[6]	+	none	>3.15%	—	05 25	—	{32}[2]
				night	—	—	—	none	none	—	—	00 25	—	{48}
			October 16	day	980	—	2 : 8[7]	trace	none	—	—	—	—	—
				night	280	1026	—	—	—	—	—	—	—	—
			October 17	day	504	1025	—	none	none	—	—	—	—	—
				night	56	1030	—	none	none	—	14.9	—	4.3	—
			October 18	day	—	—	—	none	none	—	—	—	—	—

[1] Phosphate ratio expressed in terms of $\frac{n}{10}$ acid : $\frac{n}{10}$ base per 10 cubic centimetres.

[2] After 15 grammes of urea.

[3] After one litre of water.

[4] Six hours after 3 grammes of sodium chloride.

[5] Up to nine hours after sodium chloride.

[6] After 15 grammes of urea.

[7] After one litre of water.

[8] = 18.5%

[9] = 80%

nephritis as well as in gout, following on the work of Chauffard and Garrod. It is known that uric acid can be detected in the blood in measurable quantity long before urea assumes pathological proportions and its estimation has been found to give a very good prognostic idea of the possible onset of chronic nephritis. Especially is this so in plumbism. Hansmann, Baumann, Davis and Stevens[4] say that: "The uric acid concentration in the blood is a delicate, if not the most delicate index of renal function at our disposal."

Table I. is instructive. The average normal uric acid concentration in the blood is about two milligrammes per hundred cubic centimetres.

The routine investigation of a chronic, slowly progressive toxic nephritis is much the same as in the others mentioned, the differences being in degree rather than in kind. In this condition we are likely to find a much lowered urea concentration in the urine and a constant low level or a further lowering is of grave import. Not much information can be gathered from albumin and casts alone, but urea retention is perhaps the best prognotic test we have. An occasional blood urea estimation correlated with estimation of the urine urea will give much information.

I have said at the start that albuminuria of itself means nothing. It does not, but no research as far as I know has been done to elimi-

nate one possible factor and that is the existence in uncomplicated albuminuria of a lowered "threshold," that is a renal albuminuria analogous to renal glycosuria. The existence of such a condition in all orthostatic or adolescent cases would eliminate the possibility of there being an actual lesion in the glomerulus or tubule. We feel now that such a lesion does not exist in these cases, but I should like to be certain. What I should do, would be to estimate the blood protein during fasting and after a large nitrogen intake concurrently with a similar estimation in the urine in a large number of apparently normal controls and in these patients with albuminuria. We know that after severe exertion albuminuria occurs; this seems to indicate that a big "head" will force albumin through, or to put it another way, that there does exist a "threshold" for albumin, as for sugar and salt.

Albuminuria with cylindruria is a more complicated problem. I frequently see this condition and I cannot help thinking that it has significance. In all the cases I see with cylindruria albumin is always present in the urine. I should like to hear the question discussed.

By the courtesy of Dr. Eustace Russell I have been able to investigate two cases of nephritis. The results are shown in Table II.

Commentary.

1. Patient T. has a chronic progressive nephritis. There is a history of scarlatina in childhood.

Patient M.'s urine contains albumin only under strain of exertion or occasional infection of any kind, such as influenza. It is a question whether his toxin is non-specific.

2 Both patients show variations in their acid-base-phosphate ratio, especially a definite decrease after diuresis, just as shown by Calvert, Mayrs and Milroy.[3] It thus seems that excessive diuresis would tend to lower the alkaline reserve (Leathes's method[5]).

3. Both patients concentrate urea, the one (M.) well, the other (T.) moderately well.

4. Both patients show a moderate degree of specific gravity fixation.

5. Both patients excrete chlorides. Patient T. was given three grammes of sodium chloride by mouth and excreted chloride in excess of that amount in six hours, chloride excretion returning to patients' normal amount after that period (Volhard method).

6. Uric acid is present in the blood of both patients in excess. Patient M. is interesting as showing a high concentration even in the absence of albumin and casts from the urine. This finding modifies the prognosis considerably. The uric acid was estimated by Benedict's method.[6]

7. The prognosis in both cases is bad, better in the case of M. than in that of T. in regard to the probable period of survival.

8. Both patients were on a fixed diet for the whole of the investigation, the nitrogenous and purin-producing elements being constant.

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VICIOUS CIRCLES IN THE PSYCHOSES.[1]

By JOHN BOSTOCK, M.B., B.S. (Lond.), D.P.M.,
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MENTAL disease is a subject which can be regarded from many points of view. In looking at it with the idea of finding examples of "vicious circles" one must first make clear what is meant by this term.

I would regard man much as a lighthouse, which stand four-square to the elements, fighting a lonely battle against their onslaught. You will find many such up and down the coasts, forsaken, derelict; they have not been able to weather the storms. The reason for this may be one of two, extrinsic or intrinsic. In the former the storms mave have been too strong; in the latter the fabric too weak. Most likely it was the result of a vicious circle; the faulty construction of part led to decay of the whole, the sea entering corroded still further; cracks ensued in other parts of the structure; the waves and the atmosphere influenced these and extended the rot. Finally the fabric became so weakened that a gale caused its final ruin. The point I would emphasize is that the particular storm could not have produced the destruction, if the previous vicious circles had not permitted this process.

In the same way man does not usually go under through one particular stress or the failure of any one particular organ, but through an increasing chain of cause and effect, between mind and body, between viscus and viscus, which gradually weaken his resistance. This, of course, is not a novel conception. Maudsley more than fifty years ago writing on the reason why a man went insane, put it thus:

> The uncertainty springs from the fact that in the great majority of cases there is a concurrence of conditions, not one single effective cause. What we want to have laid bare is the conspiracy of conditions, internal and external, by which a mental shock, inoperative in one case, has had such serious consequences in another. If all the circumstances, internal and external, were duly scanned and weighed, it would be found that there is no accident in madness, the disease whatever form it may take, by whatsoever complex concurrence of conditions, or by how many successive links of causation it might be generated, would be traceable as the

[1] The patients whose conditions are described were shown at a clinical demonstration held at Broughton Hall on September 24, 1924.

inevitable consequence of certain antecedents, as plainly as the explosion of gunpowder may be traced to its causes, whether the train of events of which it is the issue, be long or short. The germs of insanity are sometime latent in the foundation of the character and the final outbreak is the explosion of a long chain of antecedent preparations.

Since Maudsley's time the links of his chain have been so duplicated and triplicated that it is exceedingly difficult to orientate oneself in a tangle of which each strand is said to be vital. The great defect is variously stated to lie in sexual conflict, other conflicts, organ deficiency, endocrine disturbance, focal infection, defects in personality and so forth; everyone of which is the rallying point for a separate school of thought. The truth, I believe, to lie in all of them. They are each in a sense causal, but one system cannot be upset without having an effect on its neighbours. Hence if there is a search made for a particular disturbance, such as sexual perversion, it will be found, but equally so if an outlook is kept for aberrant personality trends or signs of focal sepsis, any of the latter may be discovered. The reason for this is that thanks to action and interaction, the various systems work in a vicious circle. It will be my endeavour by means of some cases to show roughly the interworking of these plots and counterplots which result in man losing his stability of mind.

Melancholia.

CASE I.—The patient was born in 1883. The family history showed that both his mother and sister were in a mental hospital. His upbringing was of a poor order; educated by a sister he left school at the age of twelve years. He then went to work in the building trade and later in the newspaper business and finally in a public department. He was always ambitious, had an urge to do great things, but was prevented by his poor scholarship; thus his studies of electrical work were made almost impossible by his poor knowledge of Euclid and mathematics. At the age of eighteen he had typhoid fever and was in hospital for a year; later he had appendicitis. At the age of twenty-seven he married and commenced a happy marrried life which ended chiefly on account of religious differences, though he changed his own faith in order to preserve harmony at home. His mother-in-law came to live with them and this accentuated the friction. The perpetual worry upset him and made him despondent and to overcome this he took to alcoholism. He had always had a weakness towards stimulants and after two or three glasses of whisky would commence to "talk big" and would be "out of himself." It was an unhappy life, though he tried to make the best of it. The climax came in 1917, when having risen to a high position in trade union circles, he was forced by force of circumstances and a mistaken sense of honour to support the men in what he knew to be a hopeless cause. The crash came; he lost his position and became profoundly depressed.

During this time he drank to excess, which caused a final domestic breach and he eventually joined the army. He has since lived with his wife only for a short period of three months. After a couple of years he commenced work as an electrical crane-driver and found the struggle to learn the mathematics of the mechanism very exhausting. He obtained, however, a first class ticket. Three years ago there was more trouble. While intoxicated he assaulted his sister-in-law and interfered with his own children. After this he cut his throat in a fit of remorse and was admitted to hospital. He was well orientated, but showed great depression and made several more attempts at suicide. He finally recovered sufficiently to be discharged in 1922. Having been again depressed he had another quarrel with his wife and she took proceedings on the grounds of desertion. An order for support was made out. He met the payments for six months, but later was unable to do so. This worried him and finally he returned to hospital in a very depressed condition and complained of his inability to sleep.

Physically he had well developed arterio-sclerosis and cardiac hypertrophy. His systolic blood pressure was 140 millimetres of mercury and his diastolic pressure was 80 millimetres. The skin reaction showed tendency to dermatographia. All reflexes were normal. His serum did not react to the Wassermann test.

The vicious circle in this man's case commences with a bad family history. Apart from the probability of inferior germ plasm, the suggestive effect will follow him through life. He has a characteristic manic-depressive temperament, a restless disposition and an insatiable ambition or urge to do great thtings; his inferior education and lack of opportunities limit his possibilities. This leads to his downfall; he fails in his domestic and trade life. His hereditary instability predisposes to pathological inebriety and completes the ruin. Sleeplesseness, another malign symptom, has a bad influence. Morbid depression eventually ensues; whichever way he turns, he places himself in difficulties and cannot surmount them.

His long illness from enteric fever, his appendicitis, his arterio-sclerosis doubtless have played their part in the circle, leading to inefficiency; they will have made success more difficult.

Hypochondriasis.

CASE II.—A male, aged sixty-nine years, was first admitted in 1917 with melancholia. It was then remarked that hypochondriasis was a prominent feature. This has persisted. He relates with emphasis a string of symptoms that embrace the major part of his anatomy. He is never free from the introspective study of his aches and pains. As one medical officer phrased it: "He is in the throes of a violent cold and he cannot even raise a cough."

The persistence of his symptoms with the vigorous outpouring of his complaints, the clear intellectual capacity in spite of a rather low intelligence quotient, the absence of hallucinations might well raise his mental condition to the aristocracy of the psychoses "paranoia."

So far as can be ascertained his family showed no sign of mental disturbance. His personality must be classed as of the tender variety and he had an undue fixation on his mother. Even as a young man he was ego-centric; he prided himself on his immense strength. He would go without thick underwear in winter to prove his hardiness. There was an inordinate pride in his own accomplishments and position.

In the 'eighties he emigrated to Australia and after a temporary financial reverse, he did well in his business. He was a moderate drinker, at times a teetotaler and was a non-smoker. About twenty years ago he commenced to speculate in mining and racehorses and lost everything; he then became irritable and violent. Later a succession of railway positions was terminated by a change in the department policy. This reverse was the final straw and he has done nothing since. On the physical side his weight in England was fifty kilograms (eight stone); in 1905 one hundred and forty kilograms and in 1910 over eighty-two kilograms. About twenty years ago when he commenced to lose weight he began to be always lazy, listless and without energy. There was obstinate constipation and the necessity for constant purgation.

The physical examination reveals an extraordinary amount of visceroptosis. He has dysuria, cystitis and albuminuria. There is valvular heart disease, signs of myocardial failure, such as an irregular rhythm. The blood yielded a positive Wassermann reaction. In a word he has abundant cause for many legitimate aches and pains.

There are here involved two very definite vicious circles. The psychic commences with an ego-centric

personality; consequent pride in his own judgement leads to faulty decisions and failure results. Later he is the victim of circumstance. He reacts to his environment by a phase of depression the form of which is decided by the second circle. A metabolic disturbance leading to a decrease in weight, visceroptosis and subsequent derangement of all the abdominal functions fixes his attention on the ensuing symptoms. Heart disease, albuminuria and cystitis complete the process.

Melancholia.

CASE III.—This patient, aged forty-nine years, was admitted in January, 1924, in a condition of melancholia after having made several suicidal attempts. She was profoundly depressed, said she burnt herself as she could not face the consequences. She considered that she was sinful. She had hallucinations of hearing at night, but would not specify them. Her orientation, perception, recognition and so forth were good.

Since then she has not improved. She thinks in terms of suicide and has to be kept in close observation. The delusion which troubles her most, is that she has lost all her power and cannot lift the "slightest little thing."

As a child she had an unhappy adolescence; she is said to have been below average at school and to have been somewhat bad tempered. Marriage occurred in 1908 and she travelled to South Africa and thence to Australia. She appears to have had a happy married life, except on certain lines. The only child was born twelve years after marriage in a difficult labour, but was followed by a prolapse of the uterus, after which marital intercourse became a burden. To her great regret the child died at the age of nine months. She had the maternal instinct well developed and later took a State child, but had to return it on account of ill-health. Her life was unduly lonely on account of her husband's frequent absence on business.

Three years ago a series of illnesses occurred, influenza developed into pleurisy and pneumonia, followed by hæmaturia, heart failure and dropsy. Later the finding of a small breast tumour depressed her as she feared that an operation would be necessary. Eighteen months later she removed to another locality, where she knew no one and complained of the solitude. Blasting operations were in progress close to the house and this frightened her and she became convinced that people were listening to her conversation. As evidence of the state of her mind at this time, she was found burning a cane and when asked the reason, said that it might be used in evidence against her, as she had used it to beat the child with when it was naughty.

In July, 1923, on account of suicidal attempts she was brought to Broughton Hall.

The beginning of her psychosis was laid in the unhappy child life and an ingrained lack of emotional control. Later unsatisfied maternal urge, marital difficulties and solitude, accelerated by change of location acted in a vicious circle. Nevertheless nothing might have happened but for severe physical illness which adversely affected her health and tended towards depression. To this may now be added the climacteric as menstruation ceased a year ago. She has definite hypothyreoidism and arteriosclerosis. Her systolic blood pressure is 155 millimetres, her diastolic pressure 100 millimetres of mercury and her arteries are thickened.

Dementia Præcox.

CASE IV.—The patient was born in 1898. His father was a drunkard. Adolescence was a struggle, the two brothers having to fend for themselves while a somewhat harsh mother gained a livelihood. He became an electrician and finding work difficult to obtain in Scotland, he took passage to Australia two years ago. At first he found employment, but later was put off through no fault of his own. Later he found his way to a mental hospital in Victoria and finally was admitted to Callan Park in May of this year, after having attempted to commit suicide. At that time he had insight into his condition and was accessible, but latterly he has become sullen and reticent. He is bothered by vivid hallucinations and will suddenly turn and shout to them to leave him alone. He will not specify their nature. He shows no real dementia, as his intelligence as evidenced by the Simon-Binet test is that of a superior adult.

Upon his admission he had severe anæmia, the number of red blood cells being 3,600,000, of white blood cells 12,500, of which polymorpho-nuclear leucocytes were 63%, large lymphocytes 8%, small lymphocytes 24½%, eosinophile cells 1%, basophile cells ½% and transitional cells 3%.

Many factors are present in this psychosis. The first are the bad family history and the indifferent upbringing. He appears to have had a schizophrenic disposition; he suffered from dour moods, "would flash up in a minute if spoken to" or otherwise be sulky or again day-dreaming. He was reserved and had a shut-in disposition. It was hardly the temperament to stand up to stresses.

Resulting from his immigration we find a number of conflicts. His aunt said it would kill her and sure enough she died shortly after he left. He became enamoured of a girl on the boat and later on saw the photograph of another man on her table. He lost his employment. He became acutely homesick and regretted having left the old country. Finally he was given the chance to be repatriated, but refused to go. This worried him afterwards and he would repeat: "Oh, that I had never left Scotland!" At other times reminiscences of his former life would trouble him and he could hear his aunt speaking: "You have killed your auntie." Finally his obsessions and his affective state interfered with his sleep; he became more depressed and decided to take his own life. This cycle of events found a suitable parallel in his physical condition.

The bowel was very overloaded, the skin was hypo-æsthetic. There was considerable thyreoid deficiency as shown by his tolerance of the extract, large doses of which have been given without rise of temperature or pulse rate.

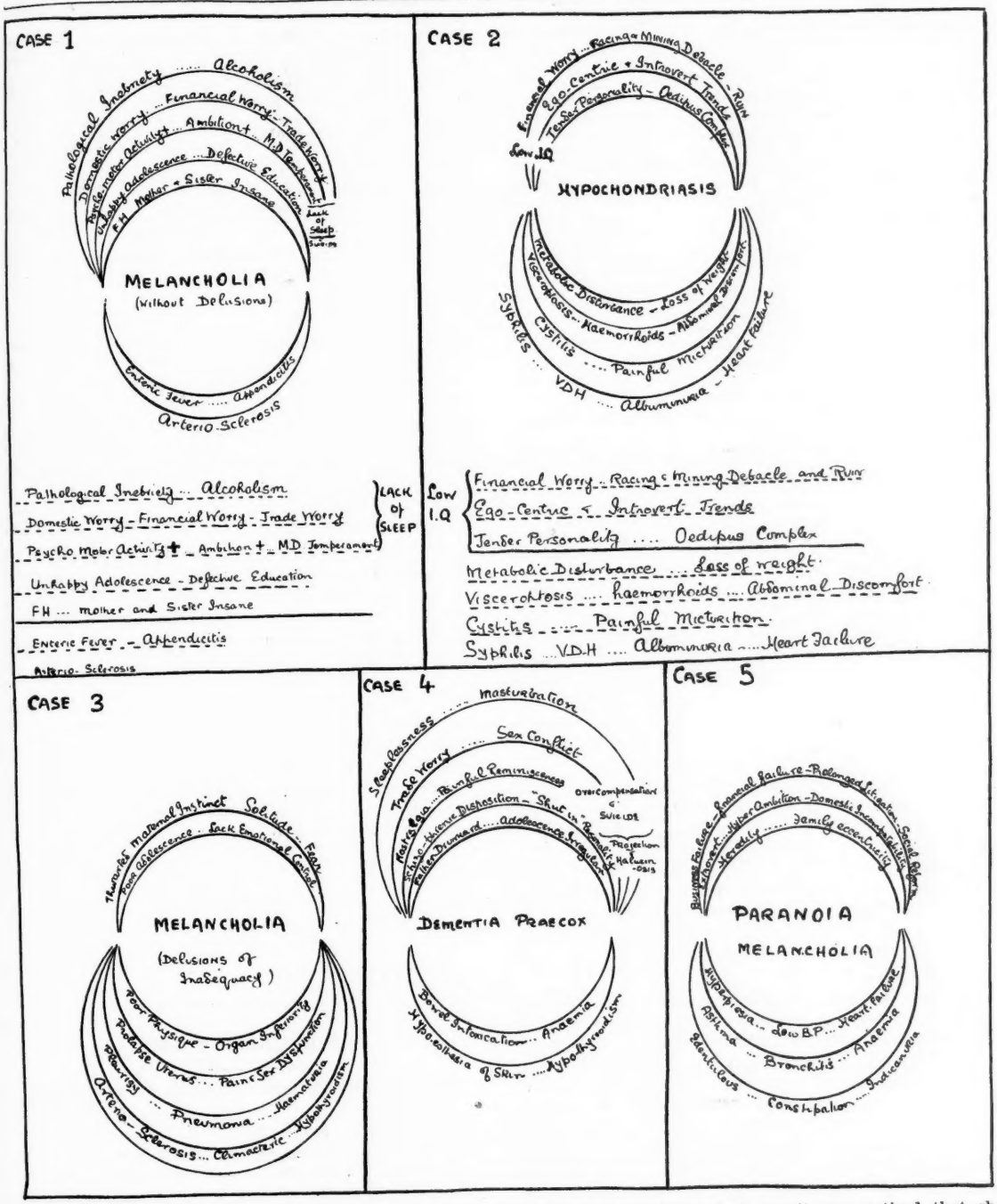
To deal with the past is, of course, a matter of conjecture, but in my opinion, could we have severed these vicious circles at an early date, that is childhood or even before he left his home for Australia and at the same time have given him a more harmonious environment, we might have saved him from his psychosis.

Paranoid Reaction.

CASE V.—This patient, aged fifty-seven years, was admitted two years ago with a provisional diagnosis of paranoia. She was discharged two months later, but returned subsequently with her paranoid reaction much in evidence. In six months she has lost this and become an agitated melancholic, with extreme ideas of unworthiness and a very depressed affect.

The first factor of which we are cognizant, is an eccentric father. Her temperament was that of the extrovert. She was capable, busy and extremely ambitious. From what I have been able to gather the means to her end were not always strictly legitimate. Her husband's financial embarrassment in early married life served only to whet her strivings, though at the same time it showed her eccentricity. For years she lived in the same house with him without speaking and eventually tried to rid herself of his presence by shooting him in the back.

She went into business and made a great success of a patent. This was to have been bought by the govern-


CASE 1
Pathological Inebriety ..... Alcoholism
Domestic Worry ... Financial Worry - Trade Worry
Psycho-motor Activity? ... Ambition + ... M.D. Temperament
Unhappy Adolescence ... Defective Education
F.H. Mother - Sister Insane
Lack of Sleep
MELANCHOLIA
(Without Delusions)
Enteric Fever ..... Appendicitis
Arterio-Sclerosis
CASE 2
Financial Worry ... Racing & Mining Debacle - Ruin
Ego-Centric + Introvert Trends
Tender Personality - Oedipus Complex
Low I.Q.
HYPOCHONDRIASIS
Metabolic Disturbance - Loss of Weight
Visceroptosis ... Haemorrhoids - Abdominal Discomfort
Cystitis .... Painful Micturition
Syphilis ... V.D.H .... Albuminuria - Heart Failure
CASE 3
Thwarted Maternal Instinct Solitude ... Fear
Poor Adolescence - Lack Emotional Contact
MELANCHOLIA
(Delusions of Inadequacy)
Poor Physique - Organ Inferiority
Prolapse Uterus ... Pains Sex Dysfunction
Pleurisy ... Pneumonia ... Haematuria
Arterio-Sclerosis ... Climacteric ... Hypothyroidism
CASE 4
Masturbation
Schizophrenic ..... Sex Conflict
Inadequate Ego ... Painful Reminiscences
Mother-Intense Disposition - "Shut in" Personality
Father Drunkard ... Adolescence Irregular
Over-compensation & Suicide
Projection Hallucinosis
DEMENTIA PRAECOX
Bowel Intoxication ... Anaemia
Hypoaesthesia of Skin ... Hypothyroidism
CASE 5
Business Failure - Financial Failure - Prolonged Litigation - Social Ostracism
Unhappy Childhood ... Hyper Ambition - Domestic Incompatibility
Heredity ..... Family eccentricity
PARANOIA
MELANCHOLIA
Dyspepsia ... Low B.P. ... Heart Failure
Asthma ... Bronchitis ... Anaemia
Edentulous ... Constipation ... Intoxaemia

Pathological Inebriety ... Alcoholism
Domestic Worry - Financial Worry - Trade Worry
Psycho Motor Activity + - Ambition + - M.D. Temperament
Unhappy Adolescence - Defective Education
F.H. ... mother and Sister Insane
} LACK OF SLEEP

Enteric Fever - Appendicitis
Arterio-Sclerosis

Low I.Q. { Financial Worry - Racing & Mining Debacle and Ruin
Ego-Centric & Introvert Trends
Tender Personality .... Oedipus Complex

Metabolic Disturbance .... Loss of weight.
Visceroptosis .... haemorrhoids .... Abdominal Discomfort.
Cystitis ..... Painful Micturition.
Syphilis ... V.D.H .... Albuminuria .... Heart Failure

ment, but it did not materialize. There appears to have been some reason for her claim for damages which were assessed at a high figure, but the suit failed. Since then she has woven a quasi-delusional system of delusional ideas into her intellectual system, of conspiracies, vested interests, personal spite and jealousy on the part of her enemies. For years she fought her hopeless fight by litigation and in a characteristic paranoid manner refused to admit defeat. A by-path of her virile extrovert personality was seen by her championship of social reforms. In these she took a prominent part.

Upon her previous admission it was noticed that she had phases when she was diffuse and rambling, showed agitation and auditory hallucinations. Since then the psychosis has changed. In the place of paranoia the scene is one of melancholia. She believes she is in some way committing a great wrong with regard to State nationalization.

She is no longer the vigorous fighter against grievances, but has made a complete *volt face* and has become a guilty conspirator, full of strange fears and fancies. She is obsessed by delusions of unworthiness which are buttressed

by hallucinations of hearing. She has over-compensated, projected, rationalized ideas respecting them.

On the physical side asthma and bronchitis which have troubled her for years, are now worse. She is very anæmic. The red blood corpuscles number 4,000,000 and the leucocytes 5,625. There are 50% polymorpho-nuclear leucocytes, 7% large lymphocytes, 23% small lymphocytes, 9% eosinophile cells, 3% eosinophile myelocytes and 5% transitional cells. She has complete dentures, is obstinately constipated and has persistent indicanuria. Most remarkable of all her systolic and diastolic blood pressures which in 1922 were 220 millimetres and 140 millimetres respectively, are now only 140 and 100 millimetres. She has valvular heart disease. Her physical condition has greatly deteriorated in the last few months.

In the face of these facts I would suggest we are dealing with two main vicious circles, the one psychic, is based on eccentricity, strivings and urges; ambition which accomplished defeat, terminates in paranoia. The second depending on her physical state includes the asthma, bronchitis, anæmia, hyperpiesis and failing circulation. These conduce towards a condition of affective depression with I am afraid some dementia. It is the latter vicious circle which now holds the field.

With regard to the schemata that have been used to illustrate these examples of mental diseases, they must be regarded as mere brief summaries or chapter headings.

The ideal case history would require a synthesis which would comprise every minute of the patient's life. It would show all the complex interactions of cell and cell, between organ and organ. The number of such combinations is infinite. Until we know more of them the pursuit of medicine must be pioneering in an unknown land. To take a concrete example. How often do we find a classical case of general paralysis of the insane? The answer is: rarely. Yet here we have a well known and comparatively simple organism, which can be carefully examined in the laboratory and its properties accurately determined. How is it that in one individual it dies out, in another the onset of symptoms occur in a relatively short period or an extremely long one or again how is it that the form of the psychosis may be roughly euphoria, dementia, tabes and so on? Why does not the spirchæte attack each individual in exactly the same way?

One might reply to this by ascribing animistic properties to the protozoon and endowing it with different tastes and affinities, but more likely its behaviour is dependent upon the varying chemical and other activities in the host. The fact that there is such a number of variations in its *modus operandi*, must surely denote a corresponding multiplicity of reactions in its environment.

Were these factors known it would be possible to postulate that an infection with the spirochætes must inevitably result in a psychosis of a given type. In all probability the number of facts which would be necessary for the formulation of such a prognosis, would be extremely great and they would embrace every activity of the individual. In the same way every other psychosis is a complex.

The value therefore of these schemata is not in their completeness, for they are very incomplete, but in suggesting in a graphic manner the interplay of many factors and their vicious circles. It is an idea which must be ever before our minds. To consider any clinical entity apart from its surroundings, is as heinous an offence, as it is to wrench a sentence out of its printed setting, without mention of that which precedes or follows. Yet both are by no means rare occurrences.

By a method such as this, one is reminded of the context. Since writing the foregoing, criticism has been made that one of the essential principles of a circle is that it begins and ends in the same place. This is a valid argument and can only be countered by stating that the term "vicious circle," as adopted by many writers in medicine, is a symbol which has no precise mathematical meaning. One is permitted to use it, even though the two ends come but loosely into juxtraposition or be brought together through an intermediary. To meet the objection, however, the term vicious "strata" might well be coined. Looking upon the individual as a collection of layers, psychic and organic, the presence of faults or weaknesses might damage the stability of the whole. The first two cases have been diagramatically represented in this manner.

At the risk of indulging perhaps too freely in metaphor, I would compare one of these patients to a man who finds himself in a morass. Each step he takes leads to further difficulties. He commences to sink deeper and deeper. His outlook or prognosis will depend largely upon the distance he has taken from *terra firma*. The treatment will depend on the finding for him of a foothold. We must endeavour to reach him with the means at our disposal. It may be a ladder of persuasion, a rope of suggestion, a scantling of organo-therapy or a plank of dietetics, hygiene or medication. Unhappily, many will sink beyond our aid.

Acknowledgment.

I am indebted to Dr. J. A. L. Wallace, Medical Superintendent of the Mental Hospital, Callan Park, for permission to publish an account of these cases.

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Reports of Cases.

THREE CASES OF EPITHELIOMA OF THE PENIS.[1]

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I WISH to report three cases of epithelioma of the penis mainly because of the comparative rarity of this condition. All three patients were admitted to and treated in the Adelaide Hospital within a period of two months.

CASE I.

History.

L.O'T., aged forty-one years, single, complained of a lump on his penis, commencing as a small lump under

[1] Read at a meeting of the South Australian Branch of the British Medical Association on September 25, 1924.

the prepuce ten years before admission to hospital. It was not painful and there was no discharge until six weeks before admission, when it became painful and began to discharge. It had a very foul odour.

Physical Condition.

The *glans penis* was entirely replaced by a large, fungating, irregular, hard cauliflower tumour extending towards the base of the penis with a hard, irregular, infiltrating margin. There was a thin discharge from the ulcerating surface which surface also showed slight signs of bleeding readily. There was slight excoriation of the skin. There were hard, discrete, enlarged, palpable glands in both groins. These were neither tender nor inflamed.

Operation.

The penis was amputated, a long dorsal flap being made. A new meatus was formed from the urethra. The glands were removed from both groins and drainage tubes were inserted.

Remarks.

The patient had no difficulty from inability to pass water. Microscopical examination revealed an infiltrating epithelioma of a type that would probably not give rise to early metastases. There was lymphatic hyperplasia of the glands, but no deposits. A peri-urethral abscess of recent formation was present.

CASE II.

History.

W.B., aged sixty-three years, married, complained of a lump on the penis which first appeared three years before admission to hospital. He did not think it serious and thought it would get better. It got worse. He saw a doctor three weeks before admission who removed a piece for section which proved to be epitheliomatous. It was painful at the start, but not latter. It had a discharge at first whitish, then fawn colour.

Physical Condition.

There was an indurated deep ulceration under the prepuce and on the *glans penis* beneath the meatus and on the frenum. There were hard, painless, enlarged glands in each groin.

Operation.

The penis was amputated, a long ventral and short dorsal flap being made about 2.5 centimetres from the base. A new meatus was made by splitting the urethra and suturing it through the flap. The inguinal glands were dissected out from both sides and drainage tubes inserted.

Progress.

Urine was passed without difficulty twenty hours after operation. The patient has been able to micturate without much inconvenience through the penile stump. After walking around some œdema of the right leg appeared; this is becoming less. The histological examination of the section of the glands did not reveal any epitheliomatous deposits.

CASE III.

History.

J.C., a Maltese, aged fifty-one years, married, stated that a lump appeared on his penis three years before admission to hospital. It increased in size, but was not painful. He lost a considerable amount of weight before admission.

Physical Condition.

There was a large papillomatous growth (3.75 centimetres by 5 centimetres) involving the *glans penis* and the prepuce. It was red in colour with a thin sero-purulent discharge and a foul odour. It was not tender. It was hard and did not seem to be infiltrating the penis. No glandular involvement was detected. A piece was removed for section. The pathologist reported that the lesion was non-malignant.

Operation.

The penis was amputated by means of a long dorsal flap and a new meatus was formed. The glands were not removed.

A further microscopical report of the growth was made and the pathologist reported: "An epithelioma of papillomatous type and although invasive would not tend to give rise to early metastases."

Progress.

The patient passed his water without difficulty fifteen hours after operation and has had no difficulty since.

Comment.

The outstanding features of these cases seem to be (i.) the presence of a painless, fungating growth involving the glans and prepuce and locally infiltrating the penis, (ii.) that a space of years had elapsed before the growth caused sufficient inconvenience to the patient for him to see a doctor, (iii.) the absence of metastatic deposits in the neighbouring glands, which fact, I take it, means a more favourable prognosis and (iv.) foul odour arising from the growth.

Acknowledgment.

In concluding, I wish to thank Drs. Newland and Corbin for permitting me to report these cases.

INTESTINAL OBSTRUCTION FROM GALL STONES.

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REPORTS of two cases of intestinal obstruction due to gall stones in a recent number of *The British Journal of Surgery* with a note as to the rarity of the condition, encourage me to put the following case on record.

MRS. M.A.F., *ætatis* fifty-one years, called me to see her one morning on account of agonizing spasmodic pain in the upper segment of the abdomen about the centre, which had begun at four o'clock in the morning of that day. With the onset of the pain she had vomited her previous two meals and later some bile. Vomiting was still frequent when I saw her. A hypodermic injection of 0.015 gramme (a quarter of a grain) of morphine relieved her condition and she then considered that her trouble was over.

Next morning the pain began again slightly, but she did not think it necessary to send for me till the afternoon when she was obviously worse. The pain had the same character as on the previous day, but in addition there was general tenderness over the centre of the abdomen, but no special or localized rigidity. Nothing was to be made out in the region of the gall bladder which had all along been under suspicion because of earlier attacks of gall stone colic. The patient volunteered the information that the pain was not in the least like that experienced in these past attacks. Incidentally it is of interest to note that she had steadily refused operation for these and had finally gone to a pharmacist who appears to have given her a large dose of olive oil, as she told me that she had then passed many "rounded stones" *per rectum* and following this treatment had had no trouble with her gall bladder for the space of two years, that is up to the time when I saw her. At six o'clock in the evening of that day the vomit had become frequent and foul and the bowels were confined, though not absolutely. The pulse had risen from 60 to 90 and the temperature from 36.9° C. to 37.6° C. (98.4° F. to 99.6° F.). A diagnosis of probable intestinal obstruction of unknown origin was made and she was operated on about nine o'clock that night.

A large gall stone was found blocking the lumen of the jejunum approximately about one metre from the duodenum. A longitudinal incision on the anti-mesenteric border of the gut was used for the extraction of the stone and this was then sewn in two layers. But this in result so narrowed the lumen of the gut that I felt that a lateral entero-anastomosis was a necessity to avoid a continuance of the obstruction. Palpation then revealed a shrunken gall bladder with another smaller stone deep in this. On the incision being extended, the gall bladder was found densely adherent to the greater curvature of the stomach near the pylorus. An attempt at cholecystectomy led to the opening up of a stoma which the larger

stone had formed by ulceration; this process had culminated in its expulsion into the stomach. As time was pressing, the second stone was removed from the cystic duct, the fundus of the gall bladder was cut away and the pathological gastro-cholestostomy was repaired. A stab drain was left in the right loin. The patient left the table in good condition with a pulse of 120, but died within thirty-two hours probably from the intense toxæmia of the high obstruction as vomiting was very little in evidence after the operation.

The dimensions of the larger gall stone were 3.2 by 2.2 by 2.7 centimetres.

Statistics go to show that intestinal obstruction due to gall stones may be considered a very serious complaint. During the twenty years up to 1907 four out of five patients with gall stone impaction, died at Saint Thomas's Hospital. Rendle Short gives the mortality as about 57%, but points out that these statistics are old.

While many factors must contribute to the seriousness of this condition, a dominant one would seem to be that the obstruction is likely to be high in the bowel; as, if the stone is small enough to pass the first meter or two of jejunum easily, there seems to be no real reason why it should not finish its course safely. And experience justifies the generalization that an obstruction high in the alimentary canal from whatever cause, is always a more serious thing than one lower down. Compare the comparative freedom in the early stages from urgent symptoms of a patient with a ring cancer of the sigmoid who suddenly developes a complete block.

BILATERAL HYPERTROPHY OF SEVENTH CERVICAL TRANSVERSE PROCESS WITH BILATERAL RUDIMENTARY ACCESSORY RIB.[1]

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THE patient, E.G., aged thirty-five years, was admitted to the out-patient department of the Royal North Shore Hospital on January 8, 1924. She complained of (i) pain down left arm since she was a girl and down right arm for about four years, both intermittent in character and often aggravated by minor ailments; (ii.) wasting of the ball of the left thumb; (iii.) inability to keep the arms warm in winter, particularly the left hand; (iv.) weakness in both arms, especially the left; (v.) an aggravation of all symptoms following pneumonia five months previously. The personal and family history showed nothing else of importance.

The atrophy of the left thenar eminence was very obvious and most noticeable in the *abductor pollicis*. There was also atrophy of the interossei, most noticeable in the first and becoming less towards the medial side. In the right hand there was slight atrophy in the first interosseous space and atrophy was indefinite in the other muscles.

No bony mass was palpable in the posterior triangles, the subclavian artery was not unduly prominent nor was any bruit audible. There were no pupillary changes. On admission there were no sensory changes and there did not appear to be much difference in colour between the two limbs.

Owing to the indefinite nature of the skiagram of the cervical region and there being some doubt as to possible fibrillary changes in the lower part of the trapezius, it was decided to keep the patient under observation pending installation of the new X-ray apparatus. The first report was: "Transverse process of seventh cervical vertebra enlarged, but not necessarily pathological; no osteo-arthritis."

About one month later numbness and tingling appeared in the distribution of the ulnar nerve extending as high as five centimetres above the medial condyle of the humerus in the left arm only. There has also been occasional anæsthesia in the index finger in the same arm.

The patient was put to bed for one week with the arms kept above the head, but this caused an aggravation of the pain and the feeling of coldness. It was then noticed that when the arms were held above the head with some flexion at the shoulder, the radial pulse on both sides could be obliterated. No œdema was caused in any position.

A further skiagram was now taken and the report was as follows: "Hypertrophy of transverse process of seventh cervical vertebra and rudimentary cervical rib on each side."

In *La Presse Médicale*, November 21, 1923, page 969, O. Crouzon states in a *résumé* of the subject that in seventy patients with cervical rib syndromes, fifty-six had hypertrophy of the transverse processes only, five had cervical ribs and large transverse processes, eight had rudimentary ribs with large transverse processes and only one had a cervical rib with a normal transverse process. In a further study of forty-five of these cases the condition was unilateral in twenty-five and more frequent in women. In only two instances could the rib be palpated. Sensory disturbances were shown in fifteen patients and atrophy only in ten. Circulatory disturbances were present in four patients and pupillary changes in eight.

The definite atrophy of the *abductor pollicis brevis* which is pointed out by S. A. K. Wilson as a feature of the condition though not pathognomonic, is well seen in this patient.

The aggravation of the symptoms after pneumonia is interesting. It is not uncommon for the onset of symptoms to date from an acute illness. Increased compression due to traction might thus result from a drooping of the shoulders due to a hypotonic condition of the muscles and increased pain might be caused by a toxic neuritis.

The obliteration of the radial pulse by raising the arms above the head is an unusual feature. In only 9% of the series of Crouzon was any circulatory interference found. This interference was manifested by the diminution or disappearance of the radial pulse when the arm was hung by the side. But in this patient the position was reversed, the pulse being good when the arm was hung down and becoming feebler as the arm was raised to disappear with the arm fully elevated. This could be caused by the subclavian artery being drawn against the inferior surface of the rib or compressed by a fibrous extension. The feeling of coldness was probably nervous in origin as there did not appear to be appreciable interference with the pulse with the arm in any but the elevated position.

Finally the uncommon occurrence of cervical rib as compared with hypertrophy of the transverse process, is seen in the series of Crouzon.

[1] The patient whose case is reported, was shown at a meeting of the New South Wales Branch of the British Medical Association on November 13, 1924.

Reviews.

RADIUM AND LIVING TISSUE.

DR. SIDNEY FORSDIKE'S essay on the effects of radium upon living tissues is an excellent and clearly written monograph. It is concise without being unduly condensed and yet it is comprehensive.[1] The author has put the case for radium in a broad-minded and unbiassed manner and without undue optimism. He is to be complimented.

Unfortunately it is not clear in the article on uterine hæmorrhage, whether the dosage is in terms of radium element or radium salt. Presumably the latter, as in the first part of the book he mentions doses in terms of radium sulphate. However, this is a vital point in which there should be no room for doubt.

A well chosen series of illustrations of microscopical sections indicates clearly the results of the action of radium on the tissues and there is a short explanation of each illustration in the text.

This is an excellent little book and will be very helpful to those who desire to know more of the therapeutic value of radium in malignant disease and uterine hæmorrhage and how and why it acts in a beneficial manner.

[1] "The Effects of Radium Upon Living Tissue: With Special Reference to its Use in the Treatment of Malignant Disease (Jacksonian Essay)," by Sidney Forsdike, M.D., B.S. (Lond.), F.R.C.S. (Eng.); 1923. London: H. K. Lewis and Company, Limited; Demy 8vo., pp. 72, with nine plates containing 42 illustrations. Price: 5s. net.

The Medical Journal of Australia

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Mental Hygiene in Industry.

THE medical industrial hygienist is concerned mainly with the health and safety of the worker. His work is largely preventive. Under ideal conditions he examines individuals before they are engaged by an industrial employer; he determines whether or not they are in a suitable state of bodily health to be prepared for the hazards which are associated with their labours. He also keeps a constant supervision over all persons who are engaged in the work of the establishment. Not only does he treat those who become ill or who meet with accidents, but he must see that all machinery is so protected that the possibility of accident is reduced to a minimum; he must see that the surroundings are hygienic, that the hours of work are suited to the nature of the industrial process and to the employees and that the latter are selected and placed according to the work best suited to them. It is obvious that the several types of work will be performed most satisfactorily by persons whose physical condition and mental equipment are best adapted to the particular branch of the business. A person of low mental calibre cannot possibly compete with another whose mentality is of a much higher grade in work which calls for rapidity of decision and judgement. It is the duty of the medical industrial hygienist to see that the mental equipment of an individual is sufficient for his work. To this aspect the term mental hygiene has been applied.

The subject may be regarded from the point of view of both employer and employee. The latter may be considered first, because it is with the employee that the medical practitioner is mainly concerned. An employee, engaged in work for which he is mentally unsuited, will not be happy, he will lag behind his fellows, he will require to make continuous effort which he will not be able to sustain, and as a result he will be miserable and discontented and his health may ultimately become affected, probably by a functional affection of the nervous system. It is also likely that an employee who is capable of mental effort of a high order, will find his work irksome and monotonous, if he is engaged on some task thought by him to be dull and mechanical. Regarded from the point of view of the employer mental hygiene is of interest, because it promotes harmony in his establishment and tends towards a larger output of work. It has been claimed that the employees of a large establishment may be divided from the mental standpoint into three groups. There is a small group of persons who suffer from some definite mental disturbance; there is a second group, constituting between one-third and one-half of the total number of employees, whose members are not adapted mentally to the particular occupation allotted to them. The third group is composed of individuals who with encouragement and stimulation may be made brighter and happier and who will thus become efficient.

It is not always the case that a medical practitioner acts as referee in regard to the placing of an employee when suitability dependent on mental condition has been called in question. The superintendent of a factory may decide the point at issue and he is frequently an individual who, having risen from the ranks, understands thoroughly the whole industrial process. In any case he will look at the question from the point of view of man power and work to be done. It cannot be expected that he will be able to gauge the effect of a continuous series of events on the mind of another individual. The referee may, however, be a so-called lay psychologist, but without any knowledge at all of the application of psychology to human beings in health and disease. He may understand perfectly the methods of determining intelligence quotients and so forth, but will be utterly at sea when confronted either with a malingerer or with the necessity of making a determination with regard to a person suffering from neurasthenia. Moreover, in the nature of things he would be helpless, because he would not recognize the cause and effect in regard to an employee whose nervous instability had reduced him to such a level that, for example, his digestion had become

seriously affected. It will thus be seen that the person to deal with this question is the medical practitioner with an extensive understanding of physiology, specially trained to undertake the work. He must have a sound knowledge of medicine and surgery, must be able to judge the mentality of a worker and, as has been pointed out repeatedly in these pages, he must understand the industrial process from beginning to end. Theoretical knowledge is not sufficient; he must be able to put himself in the place of the person in regard to whose condition he is asked to express an opinion.

Two further points require emphasis. In the first place the question of mental hygiene does not end with ideal conditions at the factory. However perfect these may be, a *mens sana in corpore sano* cannot exist if after working at a congenial occupation in hygienic surroundings the worker goes home to a squalid and insanitary environment. He also requires certain time for leisure and recreation. This question is of such importance that it was discussed at some length at a recent international labour conference held under the auspices of the League of Nations. The other point to be remembered is that mental hygiene is not a thing apart. It is part and parcel of industrial hygiene. It is not a catch-phrase that means nothing. It is important and merits the attention and study of medical practitioners.

Current Comment.

THE MECHANISM OF ANAPHYLAXIS.

ALTHOUGH it is over a quarter of a century ago since Charles Richet published his classical work on anaphylaxis and the amount of research that has been undertaken since that date on this subject, has been enormous, the definition of the term still varies considerably according to the views of the writers. At one time almost every biological reaction associated with some degree of shock was regarded as an anaphylactic phenomenon, whether the reaction were a manifestation of hypersensibility or not. Richet limited the term to a state of increased susceptibility induced in an animal by a sensitizing injection of a proteid substance. Rosenau and Anderson, Arthus, Otto and all the early workers adhered strictly to Richet's conception of the phenomenon and studied it only when the shock resulted from the injection of a protein in a previously sensitized animal or when the shock followed the injection of a mixture of the protein and the serum of a sensitized animal (passive anaphylaxis). It only introduces unnecessary confusion to endeavour to find a solution of the mechanism underlying the phenomenon from observations of other forms of toxic shock, no matter how closely the symptoms simulate those of anaphylaxis. It has long been known that the sensitizing dose calls forth a biological response in the form of an immune body and that the shock is the toxic symptom of a combination of this immune body and the originating protein. On the basis of this various workers have built up hypotheses to explain the mechanism. There is difference of opinion as to whether the amboceptor or immune body is sessile or free. As the characteristic symptoms of anaphylactic shock are supposed to be localized in the nervous system, in the lungs or in certain viscera, those who support the cellular response with fixed receptors, have postulated the nerve cells, the muscle cells of the bronchi or the cells of peripheral organs as the site of sensitization. Dale adduced evidence of a cellular site of the receptors. He washed out the uteri of virgin guinea pigs through the abdominal aorta and after removal of the organs and immersion in Ringer's solution, brought them into contact with horse serum. No contractions were recorded of the normal guinea pig's uterus, but prompt contraction followed of the uterus of a guinea pig previously sensitized with horse serum. Coca's experiments confirmed Dale's findings. Dr. I. L. Kritschevsky has sought during the past six years to explain the phenomena of anaphylaxis and of death as a result of a change in the degree of dispersion of the colloids of the organism. Having satisfied himself that this view is correct, he and Dr. O. G. Birger endeavour to ascertain whether the changed dispersion takes place in the colloids of the blood or in the colloids of the protoplasm of the cells.[1] For this purpose they found it necessary to remove entirely the blood of an animal and to substitute for it a crystalloidal fluid. This is impossible in warm-blooded animals, but it can be achieved in a "salt frog." In their experiments the blood was washed out through the inferior *vena cava* by means of Ringer's solution or of Ringer-Locke's solution. They found that thirty-two salt frogs out of a total of fifty-three survived the experiment for more than twenty-four hours. Those animals which survived, as a rule did not differ from normal animals immediately after the substitution of the crystalloidal fluid. In the next place they tested the effect of intravenous injections of the serum of warm-blooded animals, of the juice of *Cotyledon scheideckeri* and of an acid solution of arseno-benzol. The authors claim that these three substances produce the same symptom complex in warm-blooded animals and postulate the hypothesis that the shock mechanism is the same. In the salt frog the symptom complex induced is also the same no matter which of the three is employed. In the normal salt frog pinching of the skin with forceps calls forth an immediate and energetic start. After the poisoning the frog either does not react at all or if the

[1] *The Journal of Immunity*, September, 1924.

pinching be prolonged, it performs a feeble and clumsy leap. It soon loses its ability to leap and to turn over; later the reflexes disappear and it becomes paralysed. Death follows. The length of the survival is approximately the same in the case of poisoning with serum and with plant juice. The arseno-benzol drug appears to act more rapidly, but the authors explain this on the ground of the relatively large amount of the toxic substance.

The next series of experiments consisted in the testing of the poisonous effects of the serum, cotyledon juice and arseno-benzol drug in salt frogs whose blood has been washed out by means of crystalloidal fluid. They argue that if anaphylactic shock (and they include the plant poisoning and arseno-benzol poisoning in the same category) be a humoral phenomenon, the frogs whose blood had been replaced by a crystalloidal fluid, would not react to serum, cotyledon juice or arseno-benzol and would remain alive. Conversely they would interpret shock and death in these animals as proof that the change in the degree of dispersion of colloids takes place in the cell protoplasm. The salt frogs with crystalloidal fluid in their blood vessels died of shock as a result of the injection of each of the three substances. They therefore conclude that the shock is a cellular phenomenon. They noted, however, that the period of survival of the frogs with their own blood was considerably longer than that of the frogs with crystalloidal fluid, save when the arseno-benzol drug was employed. They suggest that the colloids of the blood offer some protection to the colloids of the cells from attack by a poison. If the poison be present in large quantity or in high concentration, this protection may be absent or weak. In order to take the evidence further they emulsified the arseno-benzol drug with serum, emulsion of a vibrio or with a suspension of some organ. The filtrate of these mixtures caused death within two days of salt frogs with crystalloidal fluid, while normal salt frogs survived from three to eight days. The arseno-benzol drug thus obeyed the same law as the other poisons in respect to the protection of colloids. On theoretical grounds the authors arrive at the conclusion that, while all their experimental data point to a cellular site of the phenomenon of what they term "metaballo-dispersion," by which they mean some form of change in the degree of the dispersion of colloids, in shock produced by serum, cotyledon juice or an arseno-benzol drug, it would be unreasonable to exclude the possibility of a similar change in the degree of dispersion of the colloids of the blood. They therefore postulate a diverting, defensive rôle on the part of the colloids of the blood.

These interesting and ingenious researches of Dr. Kritschevsky and his colleague involve at least three independent principles. The question whether or not anaphylaxis is a manifestation of the change of the degree of dispersion of certain colloids in the body by the introduction of other foreign colloids is a complex and difficult one and its discussion would occupy more space than is at present available. It would necessitate a close reference to the previous work of Dr. Kritschevsky. Suffice it to state that whether the physico-chemical change is the essential element of the anaphylactic shock or not, the introduction of one colloid into a solution or suspension of another necessarily must induce a change in the degree of dispersion of the latter. Intimately associated with this is the phenomenon of the adsorption of colloidal particles by protoplasm and also the rate of diffusion of these particles in colloidal media. The physical mechanism of the effect of the introduction of protein in a sensitized animal must be largely determined by these processes.

In the second place Dr. Kritschevsky ignores the biological element of anaphylaxis and seeks to reduce it to a process governed exclusively by chemico-physical laws. The third principle is the correlation of serum poisoning, poisoning by cotyledon juice or poisoning by arseno-benzol with the phenomenon of true anaphylaxis. It seems to be a bold step to assume that the poisoning with serum or other substances is the same as the production of shock by means of a harmless protein in an animal rendered hypersensitive by preliminary treatment. If this assumption is unjustified, the hypothesis concerning the change in the degree of dispersion of cellular or humoral colloids as an essential factor cannot be translated from the former to the latter. Further experiment is needed to establish whether the authors are right in their assumption, but in the meantime, we cannot accept their work in its entirety. That the phenomenon of anaphylaxis is to a large extent cellular appears to be proven.

LYMPHATIC PERMEATION IN MALIGNANT DISEASE.

THE question of the spread of malignant disease by lymphatic permeation or by dissemination through the blood stream has not been settled. The views of Sampson Handley, the protagonist of the permeation theory, and the work done on the subject by Piney have been discussed at various times in the pages of this journal. The report of an interesting case by Drs. Jeanneney and Mathey-Cornat deals with this subject.[1] The patient whose condition is recorded by them was a man, aged sixty-three years. A small nodule in the left costo-iliac region began to increase in size after severe trauma from the horn of a cow. Eventually the greater part of the trunk became involved and the inguinal and axillary glands became enlarged. Examination of pieces of ulcerated tissue and of the affected glands revealed the presence of sarcoma. Drs. Jeanneney and Mathey-Cornat state that the spread of the original lesion cannot be explained otherwise than by the lymphatic permeation described by Handley. They point out that the lymphatics of the region were affected even in a direction contrary to that of the normal lymph flow. Unfortunately no *post mortem* examination was made and this robs the report of a great deal of its value.

[1] *Journal de Médecine de Bordeaux et du Sud-Ouest*, August 10, 1924.

Abstracts from Current Medical Literature.

GYNÆCOLOGY AND OBSTETRICS.

Carcinoma of the Body of the Uterus.

CHARLES E. NORRIS AND M. E. VOGT (*American Journal of Obstetrics and Gynecology*, May, 1924) report a series of one hundred and fifteen cases of carcinoma of the body of the uterus. They conclude that carcinoma of the body is less frequent than in the cervix, probably because chronic cervicitis is a common lesion, whereas true chronic corporeal endometritis is relatively infrequent. Carcinoma of the fundus is a disease of advanced life. Over 70% of their series of patients were over fifty-one years of age. Child-birth plays little part in the ætiology, 26% were spinsters. Hæmorrhage and discharge are the most important symptoms. In 81% hæmorrhage was the first sign. Pain, cachexia and loss of weight generally indicate an advanced and inoperable tumour. The histological examination of tissue obtained on curettage offers almost certain means of diagnosis, even in the early stages. The Clark test which consists in the passage of a sterile sound, is of great practical value. Absence of bleeding following this test goes a great way towards excluding carcinoma. In 75% of the unsuspected case (19% of the series) the symptoms resulting from cancer were chiefly those produced by pre-existing myomata. The chief point of prognostic value is the integrity of the myometrium. The most suitable treatment is pan-hysterectomy and bilateral salpingo-oophorectomy. Post operative irradiation by radium or deep X-rays is of distinct value. Radium irradiation is the greatest comfort to the patient and prolongation of life. It offers hope of cure even in patients whose condition is too advanced for operation.

The Effect of Radio-Therapy on Future Pregnancies.

W. SCHMITT (*Klinische Wochenschrift*, July 22, 1924) states that divided opinions are held on the effect of radio-therapy in causing fœtal malformations in future pregnancies. He gives the results of nine pregnancies observed at the Gauss Clinic. The patients were treated for severe menorrhagia or for fibro-myomata. The interval between the application of the rays and conception varied from three to twenty-two months. Eight living children were born (four boys and four girls) so no conclusions could be drawn regarding the effect on sex. One miscarriage occurred at the fourth month and no abnormalities of the placenta or fœtus were noted. The rays could hardly be blamed for the miscarriage as it occurred twenty-two months after the last treatment and regular menstruation had been present for a year. The children were all of average size and weight and were not abnormal. One child died from tuberculosis at ten months; the others have been observed for over three years and manifest no changes. From a perusal of the literature the author gathered one hundred and thirty records. Eighty-six of the patients had been submitted to X-ray radiation and forty-four to radium. Thirty-six pregnancies ended in abortion, but five of these were complicated by cervical carcinoma. Eighty-one children were born, with five deaths. Of these two were syphilitic, two were premature and one manifested skull defects. In the latter case the mother had been under treatment for Hodgkin's disease.

Asphyxia Neonatorum.

E. ZANGEMEISTER (*Klinische Wochenschrift*, September 16, 1924) states that out of one thousand and fifteen infants born in the Marburg clinic in 1922-1923, two hundred and six were asphyxiated, one hundred and eighty-three slightly and twenty-three severely. The mortality was eleven, four from asphyxia combined with intra-cranial hæmorrhage and seven from pure asphyxia. The most important point in prophylaxis is to observe the fœtal heart regularly during labour. Immediately after birth the infant should be suspended by the legs to prevent aspiration of mucus and the air passages cleared. For white asphyxia he recommends rhythmic massage of the heart with the fingers. After detailing the usual methods of artificial respiration, he describes his own apparatus. This consists of a glass nasal cannula and a rubber bulb for blowing air into the lungs. He prefers oxygen to air, but recognizes that it is not practical outside hospital work.

Prophylaxis of Puerperal Fever.

R. SALOMAN ANL S. BIERINGER (*Klinische Wochenschrift*, September 16, 1924) give the results of their experiments to reduce the incidence of puerperal fever by vaginal douches of lactic acid. They recognize four groups according to the condition of the vaginal secretion. Normally there is a white fluid with scanty leucocytes and epithelial cells. Pathological leucorrhœa is yellowish and contains many leucocytes and detritus. Daily douching with half a litre of 0.5% lactic acid will convert a pathological secretion to a normal one, usually within three weeks except when there is a gonorrhœal infection. There is no return to the pathological condition. Longer time is required if there is much bruising of the vaginal walls or a severe cervical catarrh. In a series of forty-six patients it was noteworthy that sepsis did not occur. In conclusion they advocate its use for the last fortnight before labour as a prophylactic measure.

Spirochætes in Lochial Secretions.

H. ZIELKE (*Klinische Wochenschrift*, September 9, 1924) gives the results of the examination of the vaginal secretions for spirochætes of three hundred women during the puerperium. Noguchi had described three varieties, *Treponema calligyrum*, *Treponema minutum* and the commonest one *Spironema refringens*. He found that they were more frequently present in women than men. In the secretions of nineteen women spirochætes were discovered. The frequency is less than that recorded by other authors and may be explained by the fact of taking the secretion from the cervix and vaginal walls only and not from the external genitals especially round the clitoris. The secretion containing spirochætes was generally acid and very rarely neutral. The onset of hæmorrhage owing to the alkaline secretion caused the spirochætes to disappear. They attribute the fact that none were found during the first days *post partum* to this.

Treatment of Puerperal Sepsis.

G. H. SCHNEIDER (*Klinische Wochenschrift*, August 26, 1924) summarizes the results obtained at the Frankfort Women's Hospital in the treatment of puerperal sepsis with gold and copper collargol solutions. Twenty women with severe bacteriæmia were treated and three died. The majority were due to septic criminal abortions. At first small doses of two to five cubic centimetres were given, but experience showed that the correct amount was twenty cubic centimetres. As long as it was slowly injected intravenously, there have been no cases of collapse and no observed bad effects on the functions of the kidney or liver. Rigors ceased after the injections and as a rule did not recur. The injections were given at the height of the evening rise of temperature when there were no rigors and repeated if the rigors recurred. Naturally the best results were obtained when the patients were seen and treated early. In addition digitalis was given in large doses and camphor and adrenalin.

The Treatment of Post-Partum Hæmorrhage.

T. HEYNEMANN (*Klinische Wochenschrift*, August 5, 1924) gives a detailed description of the methods employed in the treatment of *post partum* hæmorrhage. When hæmorrhage is only moderate in amount the Credé method of expression coupled with the use of pituitary extract, should be employed. For severe hæmorrhage compression of the abdominal aorta is invaluable. The author gives an account of the Haselhorst clamp which consists of a metal cross bar attached to a frame and provided with a pad which can be screwed down into the abdominal wall. He claims that it is better than the Momberg tourniquet in that only the aorta and inferior *vena cava* are compressed. After application of the clamp the placenta can frequently be easily expressed. Considerable pain is felt in half an hour and the author recommends its application for twenty minutes only. Compression of the aorta is chiefly useful in the presence of uterine atony and has less effect in hæmorrhage of traumatic origin due to lacerations of the clitoris and perineum.

NEUROLOGY.

Spasmodic Torticollis.

E. C. ROSENOW (*Journal of Nervous and Mental Diseases*, January, 1924) made cultures of streptococci from the throats of a series of eight patients suffering from spasmodic torticollis. He then gave intra-cerebral injections to seventy rabbits from suspensions of this culture. Of these forty-two developed peculiar tic-like movements, ten developed rhythmic movements and twenty turning of the head. On *post mortem* examination in all instances lesions in the central nervous system were found. The conclusion is that spasmodic torticollis is often due to a streptococcus specifically affecting certain areas in the central nervous system. Appropriate treatment to rid the throat of this organism should mitigate the distress incident to the affection.

Mesencephalitis Syphilitica.

S. A. KINNIER WILSON (*Journal of Neurology and Psychopathology*, May, 1924) has previously shown that the Argyll-Robertson pupil is probably due to a relatively superficial leptomeningitis or ependymitis affecting the mid-brain. He now presents four instances of neuro-syphilis in which there was the combination of the Argyll Robertson pupil with tremor of the limbs. He argues that this combination is not merely coincidental, as held by former investigators, but that, along with its various elaborations such as oculo-motor palsy and rigidity, it indicates deeper incidence of the syphilitic process on the mid-brain, a true syphilitic mesencephalitis.

Disseminated Sclerosis.

O. VERAGUTH AND G. GUILLAIN (*Revue Neurologique*, June, 1924) opened a discussion on disseminated sclerosis at the annual meeting of the Neurological Society of Paris in May last. Veraguth said that in Switzrland one in three or four thousand of the total population suffered. Not more than 10% showed the classical triad of Charcot (nystagmus, scanning speech and intention tremor). The cases with acute onset were the most difficult to diagnose; progress alone determined their nature. In spite of assertions to the contrary slight rises of temperature occurred in disseminated sclerosis. In differential diagnosis the practitioner should guard particularly against hysteria, epilepsy, cerebral tumour and certain slow forms of myelitis and in addition to the classical triad of symptoms he should remember its polymorphism, its remissions, the absence of abdominal reflexes, the presence of dysmetria and optic atrophy. Guillain said in regard to onset that careful interrogation showed that numbers of these patients for from six months to two years previously had noticed some kind of paræsthesia, transient visual disturbance and abnormal sense of muscular fatigue. As an opening sign sudden amaurosis was not uncommon, but hemiplegia or paraplegia was rare. Analysing the symptoms he discussed motor troubles, which consisted mainly of degrees of paralysis, hypertonus and incoordination and he mentioned that intention tremor in classical form was not as common as was supposed. Exaggeration of tendon reflexes was constant. The plantar reflexes were almost always extensor and the abdominal reflexes almost always absent. Subjective complaint of paræsthesia was common, likewise bladder weakness. Of visual disorders transitory diplopia and amblyopia with or without disc changes were frequent. Argyll Robertson pupils were not found. Cochlear and vestibular disturbance showed itself as transitory deafness, vertigo, Rombergism and nystagmus. Mental changes in the form of euphoria, uncontrolled emotions or a degree of apathy were often noticed. Examination of the cerebro-spinal fluid should include the colloidal benzoin test which in Guillain's experience induced a reaction in 63% of cases. In the matter of classification he deplored the tendency to multiply the forms of disseminated sclerosis; in his opinion not more than four deserved special description: (i.) The common form ushered in by slight locomotor disturbance, paræsthesia and vertigo; (ii.) the classical form; (iii.) that beginning with sudden visual disturbance and (iv.) the purely paraplegic form. In regard to the ætiology it was pointed out that between twenty and forty years was the average age limit. Cases recorded in children were instances of error in diagnosis. The disease was apparently more common in Europe than in America. Black races and the Japanese enjoyed relative immunity. The influence of numerous ætiological factors mentioned by others, such as exposure, trauma and the like, was doubtful; as to spirochætal infection more experiments were needed before this could be taken as proved.

Psychiatry and Public Health.

LLEWELLYS F. BARKER (*American Journal of Psychiatry*, July, 1924) in an address to the American Psychiatric Association makes suggestions for solving problems upon the relations of psychiatry to public health. These two branches of medicine have much in common. They aim at the same target. Each for the fuller achievement of its purposes requires the cooperation of a host of official and voluntary agencies outside the ranks of its own professional groups. After reference to the multiple forms of mental disorder and their origin, he indicates some practical contributinos of psychiatry to public health campaigns. The great war awakened the medical profession and the public to the extraordinary and unsuspected prevalence of mental defects among the young men who were drafted. The lesson is to frame eugenic and euthenic (improvement of environment) programmes to see that the mentally superior stocks shall not be supplanted by stocks that are mentally inferior. Then education in controlling the emotions and the will is fully as important as education of the intellect. Finally, the brain if it is to functionate properly, must be protected from all kinds of injury—traumatic, toxic and infective. In this connexion they should remember the campaign against syphilis in order to reduce the incidence of general paresis, the prevention of infectious diseases in general, the removal of chronic foci of infection and measures to prevent the abuse of alcohol.

The Treatment of Cerebral Tumours by X-Rays.

G. ROUSSY, J. BOLLACK, S. LABORDE AND G. LEVY (*Revue Neurologique*, August and October, 1924) deal in two papers with the radiological treatment of cerebral tumours. In the first they discuss ordinary tumours of the brain. The authors strike a pessimistic note. Five cases of glioma are recorded in which X-ray treatment completely failed. It is pointed out that what renders this form of treatment special unreliable is the difficulty of early diagnosis and exact localization and determination of the nature of the tumour, not to mention the danger of aggravating the symptoms. Therefore until the technique of treatment by either X-rays or radium is improved, they must still rely on the usual surgical measures. The authors claim, however, that in the case of hypophyseal tumours promising results are obtained. The first case was a classical and progressive one of acromegaly, with polyuria and visual defect. The signs of acromegaly were arrested, the polyuria became diminished and the visual defect was brought to a standstill. Of four patients with the adiposo-genital syndrome, two were greatly benefiteu, the remaining two unrelieved. Of three patients manifesting the chiasmatic syndrome along with headache, treatment was very effective in two. An important point is that these basilar and extra-cerebral tumours appear to be more tolerant of irradiation than the ordinary intra-cerebral growth. Only in one case did alarming symptoms arise, namely intense headache, vomiting and so on, coupled with the appearance of albumin and polynuclear cells in the cerebro-spinal fluid, pointing to an artificially induced aseptic meningitis.

Paralysis of the Serratus Magnus.

AUGUSTE TOURNAY AND WALTER M. KRAUS (*Journal of Neurology and Psycho-Pathology*, August, 1924) record three cases of isolated paralysis of the *serratus magnus* mainly to show that cases due to infection are rare and that the carrying of heavy weights on the shoulder added to the presence of a rudimentary cervical rib, is the more usual responsible factor. The signs and symptoms of the paralysis are given in detail, but it need only be mentioned here that attention is at once directed to the paralysis by the extraordinary winging of the scapula and the difficulty in raising the arm above the horizontal level.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Coast Hospital, Little Bay, on October 9, 1924. The meeting took the form of a series of clinical demonstrations arranged by DR. R. J. MILLARD, C.M.G., the Medical Superintendent, and the members of the staff.

Pernicious Anæmia.

A female patient, aged twenty-six years, had been admitted to hospital on August 15, 1924. She had been married for eight years and had had three children. The first had died at the age of nine months of "meningitis," the second had never walked and had died at the age of three years of "paralysis," the third was alive and well and was fourteen months old. For two years the patient had suffered from "giddy turns," breathlessness and swelling of the legs. From time to time she had suffered from small yellow "ulcers" in her mouth. She had also noticed that her skin was becoming yellow. On admission the patient had been mentally dull, she had been very anæmic and the skin had a lemon yellow colour. The teeth had been very dirty and ulceration of the mucous membrane of the mouth had been present. On August 20, 1924, the erythrocytes had numbered 1,650,000 per cubic millimetre, the hæmoglobin value had been 60% and the colour index had been 1.5. Anisocytosis and poikilocytosis had been present, but no nucleated red cells had been found. Dilute hydrochloric acid had been given in doses of eight cubic centimetres (two fluid drachms) with lemon juice, sugar and water three times a day before meals. In addition she had been given a mixture containing 0.18 cubic centimetre (three minims) of *liquor arsenicalis* three times a day after food. The arsenic had been increased by 0.06 cubic centimetre daily. Charcoal powder had been given in fifteen gramme doses (half an ounce) twice a day. On August 27, 1924, the erythrocytes had numbered 2,300,000 per cubic millimetre, the hæmoglobin value had been 60% and the colour index 1.3. On August 31, 1924, the patient had been taking 0.6 cubic centimetre of *liquor arsenicalis* three times a day. Subsequently the blood examinations had yielded the following results: On September 13, 1924, erythrocytes 3,400,000, hæmoglobin value 80% and colour index 1.1; on September 22, 1924, erythrocytes 3,600,000, hæmoglobin value 80% and colour index 1.1; on October 1, 1924, erythrocytes 4,000,000, hæmoglobin value 80% and colour index 1.

A male patient, aged thirty-seven years, married, a cleaner, had first been admitted to hospital on April 6, 1922. He had at that time stated that for the previous six weeks he had been suffering from shortness of breath, muscular weakness and pain across the chest. On examination a spastic condition of the left arm and leg dating from childhood had been found. A definite degree of anæmia had been present, the eyes had reacted normally, the tongue had been clean and smooth and had had an atrophic appearance. No abnormality had been discovered in the lungs and the spleen had not been enlarged. Three enlarged glands had been present in the right side of the neck and others had been present in the right axilla. Both chains of glands posterior to the sterno-mastoid muscle had been enlarged. The apex beat of the heart had been in the fifth intercostal space in the nipple line. The results of successive blood examinations are set out in the accompanying table.

Treatment had been carried out by the intravenous injection of arsenic and by oral administration of the same drug. No special benefit had resulted.

Achondroplasia.

A male patient, aged thirty-years, a lift attendant, had been admitted to hospital on September 24, 1924. He had complained of pain and weakness of the legs three weeks previously. He had had two similar attacks, though not so severe, eighteen months before admission. There was a history of the consumption of considerable quantities of alcohol. A left radical mastoid operation had been performed at Sydney Hospital eighteen years previously.

The patient was a typical achondroplasic dwarf. His height was 1.32 metres (four feet four inches). The cranium was large in proportion to the face. The bridge of the nose was depressed. The thorax and abdomen were well developed. The arms and legs were abnormally

TABLE SHOWING THE RESULTS OF SUCCESSIVE BLOOD EXAMINATIONS IN MALE PATIENT WITH PERNICIOUS ANÆMIA.

Date.	Erythrocytes.	Leucocytes.	Hæmoglobin Value.	Colour Index.	Remarks.
April 6, 1922	1,780,000	4,375	50%	..	Definite anisocytosis, polychromasia, punctate basophilia, poikilocytosis; two megaloblasts, one normoblast.
May 12, 1922	1,900,000	6,875	50%	..	Punctate basophilia, one megaloblast, anisocytosis.
June 20, 1922	1,300,000	5,000	60%	..	No normoblasts, one megaloblast, no alteration in film.[1]
January 12, 1923 ..	2,100,000	6,500	60%	..	[3]
February 23, 1923 ..	1,330,000	Polymorphonuclear cells 41%, lymphocytes, 59%	40%	1.6	Red cells, profound change in size and shape; polychromasia; one normoblast.[2]
January 4, 1924 ..	1,400,000		40%	1.4	[4]
February 11, 1924 ..	1,160,000		40%	1.8	
March 22, 1924 ..	1,000,000		35%	1.6	
April 11, 1924	600,000		35%	2	
April 25, 1924	840,000		30%	1.8	
May 9, 1924	1,000,000		30%—40%	1.7	
June 7, 1924	1,000,000		30%—40%	1.7	
June 25, 1924	1,260,000		40%—50%	1.8	[5]
October 7, 1924 ..	1,390,000		30%—40%	1.3	Irregularity in size and shape; some poikilocytosis and punctate basophilia; one megaloblast.

[1] Discharged July 7, 1922.
[2] Re-admitted January 3, 1923; discharged February 9, 1924.
[3] Re-admitted February 21, 1923; discharged April 6, 1924.
[4] Re-admitted January 2, 1924; discharged July 8, 1924.
[5] Re-admitted October 7, 1924.

short. Muscular power was good. The femur and humerus were short in comparison with the tibia and fibula and radius and ulna respectively. The fibulæ were longer than the tibiæ. The hands were large and square. The fingers were practically all the same length. All the joints were very lax. A certain degree of lordosis was present. The abdominal reflexes were active. Knee jerks could not be elicited. The serum had not reacted to the Wassermann test.

A series of X-ray films were shown. The prominent features of these were the shortening and irregularity of the humerus, shortening of the femur, the breadth of the second phalanges of the hand and shortness of the metacarpal bones.

Cerebral Hæmorrhage.

A male patient, aged sixty years, was suffering from cerebral hæmorrhage. On admission no history had been obtainable. He had lain in bed and apparently he had been comfortable. Conjugate deviation of the head and eyes to the right had been present. He had been able to answer questions fairly well, but could not give a rational account of his illness. He had complained of slight headache and of epigastric pains. Definite paresis of the left upper extremity had been present. Paresis and impaired sensation had been noted in the left lower extremity. The knee jerks had been present. The right plantar reflex had been flexor in type and the left had been indeterminate. The tongue had protruded in the middle line. Left facial paresis had been present. The left pupil had been larger than the right. Both had reacted to light and accommodation. The blood vessels had been thickened and tortuous. A "tinea rash" had been present on the anterior abdominal wall. The cardiac apex beat had been in the nipple line, the second sound had been accentuated at the apex, the rhythm had been regular and no murmurs had been audible. No abnormality had been detected in the lungs. After having been in hospital for some days the patient had become noisy and restless.

Carbuncle.

A male patient, aged forty-nine years, had been cut on the back of the neck by a barber three weeks before admission. A week later a boil had formed on the site. This had rapidly increased in size, had burst in several places and was discharging pus freely. Stiffness and much constitutional disturbance were present. At the time of demonstation the carbuncle extended right across the back of the neck. A stained slide taken from the discharging pus had revealed the presence of staphylococci.

Chorea.

A male patient, aged twelve years, had been admitted to hospital on October 2, 1924. An attack of rheumatic fever three years previously had been followed by heart trouble. The mother alleged that the child had been very jumpy and nervous and had dropped things. He had had the habit of swinging his arms aimlessly when walking. The child had stated that he felt well. He had been a little breathless on exertion, but had played cricket at school. On examination the decubitus had been comfortable and quiet. No tremor had been present and the tongue was clean. It had been found that the tonsils were buried and although the patient's colour was pasty, he had appeared to be well nourished. The apex beat had been 1.25 centimetres outside the nipple line and in the fifth intercostal space. A mitral systolic murmur of medium grade had been present and the rhythm had been regular. The lungs had been normal. It was pointed out that the child was intelligent and his speech was good. His actions were slightly hurried and hasty. Very slight involuntary movements were present in the forearm and hand. The cranial nerves were normal and tongue was not tremulous. The knee jerks were slightly accentuated. The plantar reflexes were flexor in type. The abdominal reflexes were active. The pupils were equal and reacted fully to light and accommodation.

Tabes Dorsalis.

A male patient, aged forty-seven years, had been admitted to hospital with a history of having noticed lack of control of the legs when walking for the previous three months. He complained of a sensation of the presence of cotton wool in the soles of his feet when walking, of shooting pains in the arms and impairment of eyesight. The patient denied having been infected by venereal disease. He was married, his wife had had one child and had had no miscarriages. The patient was anæmic and thin. His tongue was furred. His pupils were small and equal and the right reacted better to light than the left. The knee jerks were sluggish and the plantar reflex was indeterminate. Sensation to pain, heat and cold was slightly impaired. The postural sense was impaired in both legs. Slight continual fascicular contractions were present in the muscles of the legs and arms. The serum had not reacted to the Wasserman test.

Acromegaly.

A male patient, aged twenty-nine, single, a clerk, had been admitted to hospital with a history of progressive enlargement of the feet for the previous five years. This had been accompanied by pains in the joints of the feet and deformity. The patient could not purchase boots to fit him and had found it necessary to have them specially made. He had also noticed a progressive enlargement of his head so that each hat purchased had been a size larger than the last. A Wassermann test had been carried out on February 29, 1924, and a reaction had occurred. The patient had been given a full course of arsenic and mercury.

Encephalitis Lethargica.

A male patient, aged ten years, had been admitted to hospital on September 22, 1924. The patient had always been a bright, intelligent boy and had had no previous illness. Ten days before admission the patient had become extremely drowsy and would sleep all day as well as all night, if allowed to do so. When roused he had been bright and cheerful, but would go to sleep again at once. This condition had persisted after admission. Though bright and cheerful when awake, he had lost count of time and did not realize how long he had been affected. The pupils had been unequal, the right had been twice the size of the left. They had reacted to light and accommodation. No diplopia or strabismus had been present. The tongue had been protruded in the middle line and there had been no tremor. There had been no evidence of lesion of the cranial nerves. The neck had not been rigid. The knee jerks had been very exaggerated. Ankle clonus had been present on both sides. There had been some rigidity of the limbs. The plantar reflex had been flexor in type and Kernig's sign had not been present. The cremasteric and abdominal reflexes had been active. There had been no impairment of sensation. Lumbar puncture had been carried out and clear cerebrospinal fluid under normal tension had been obtained containing "a few cells, apparently pus cells, and some Gram-negative cocci."

On September 24, 1924, lumbar puncture had been carried out again and nothing abnormal had been found.

On September 25, 1924, the inequality of the pupils had been reversed, the left was larger than the right. The reflexes had not been so active, but drowsiness had been the same.

On September 28, 1924, the pupils had been equal and the patient less drowsy.

On September 30, 1924, the left pupil had been much larger than the right and the patient less drowsy.

On October 2, 1924, the pupils had been equal and the patient had appeared to be fairly normal.

A male patient, aged fifteen years, had been admitted on July 28, 1924, with the history that he had been in normal health until midnight of the day before admission, when he had had an attack of vomiting and shivering. This had continued at short intervals until 7.30 o'clcok a.m. on July 28, 1924, when he had collapsed. At this stage he had been examined by a naval surgeon. His temperature had been 38.8° C. (102° F.) and his pulse had been rapid and full. The tongue had been dried and furred. The patient, restless and lying in bed with his knees drawn up, had resisted any attempt to move him. Lumbar punc-

ture had been performed at the Prince of Wales's Hospital, Randwick, and thirty cubic centimetres of fluid under increased pressure had been obtained. Examination of a slide preparation of this fluid had revealed the presence of numerous leucocytes, but no organisms.

On admission to the Coast Hospital the condition had remained unchanged. The patient had been conscious, but mentally dull, he had answered questions slowly and with faulty pronunciation. The pupils had been equal and had reacted normally. There had been some doubt as to the condition of the knee jerks. The plantar reflex had been normal. Kernig's sign had been present. The throat had appeared injected. The heart and lungs had been normal. Some muscular resistance had been noted on the right side of the abdomen. The patient had passed urnie into the bed. The leucocyte count had been 29,000 per cubic millimetre.

On July 29, 1924, the condition had been unchanged. Lumbar puncture had been performed and forty-five cubic centimetres of turbid fluid, containing numerous pus cells, but no organisms, had been withdrawn. The fluid had been sterile on culture.

On July 30, 1924, the patient had been brighter mentally. The organic reflexes had been normal. Lumbar puncture had been performed and twenty-five cubic centimetres of turbid fluid had been withdrawn. No organisms had been found in this fluid and it had been sterile on culture.

On July 31, 1924, the patient's temperature had been normal for the first time during the illness and from this onwards his condition had steadily improved. Lumbar puncture had yielded fifteen cubic centimetres of almost clear fluid.

On August 15, 1924, the patient had complained of headache and after an interval of normality of almost two weeks the temperature had risen to 39.4° C. (103° F.). By lumbar puncture fifteen cubic centimetres of clear fluid under slightly increased pressure had been withdrawn.

On September 10, 1924, owing to the return of headache and slight mental dulness another lumbar puncture had been performed and fifteen cubic centimetres of fluid had been withdrawn. The fluid had been under slightly increased pressure and of a ground glass appearance.

At the time of demonstration the patient's condition had gradually improved, but he was not normal. He complained of slight headache and giddiness and had a "heavy look." His gait was somewhat unsteady. The pupils reacted sluggishly to light and the knee jerks were definitely exaggerated.

Dermatitis Repens.

A female patient, aged sixty-five years, a married woman, had been admitted to hospital on July 3, 1924. Two years previously she had suffered from acute inflammation of the left ring finger. This had resembled a whitlow. Shortly afterwards a similar affection of the right ring finger had occurred and later the other fingers had become similarly affected and the finger nails were shed. The patient had obtained medical treatment within one month of the commencement of the ailment. She had been treated for ten months at the Royal Prince Alfred Hospital and for four months by a skin specialist in Macquarie Street. Temporary arrest only had resulted. Just prior to admission to the Coast Hospital the lesion had spread to the right elbow, the left axilla, the neck, both knees, the right instep and the toes. On admission the palms of the hands had been sodden and ulcerated, the fingers excoriated and the nails partly shed with pus beneath them. Pustules and crusts had been present on an inflamed area in the neck and the left axilla, right elbow, both knees and the toes of both feet had been similarly affected. An effort had been made to isolate *Tinea* in the lesions on several occasions, but without result. The provisional diagnosis of *Tinea albigena* had been made. Various forms of treatment had been adopted. Baths and compresses of a one in ten thousand solution of perchloride of mercury to the hands and fingers and the application of an ointment containing perchloride of mercury, carbolic acid and oxide of zinc had produced no effect on the lesions after a period of six weeks. No definite improvement had resulted from the application for six weeks of an ointment containing salicylic and benzoic acids. *Unguentum diachyli* had produced some lessening of irritability.

Aphasia.

A female patient, aged sixty-two years, had been admitted to hospital on September 17, 1924. She had had five children and no miscarriages. Two days before admission she had suddenly become affected by partial aphasia which had persisted up to the time of demonstration. The result was that the patient used words other than those which were appropriate or which she intended to use. For example she said "please" instead of "thank you" and had a very limited vocabulary. She appeared to know what she wanted to say, but was unaware of her mistakes. Some arterio-sclerosis and cardiac hypertrophy were present. The systolic blood pressure on September 22, 1924, had been 170 millimetres of mercury and the diastolic 70 millimetres. Albuminuria was present and the tendon reflexes in the upper and lower limbs were exaggerated. The patient's blood had reacted to the Wassermann test.

Malignant Disease of the Liver.

A male patient, aged fifty-three years, a steward, married, had been admitted to hospital on August 4, 1924. The patient had been quite well until one year prior to admission, when he had suffered from pain in the lower portion of the abdomen accompanied by constipation. He had been treated medically and had remained at work until three months prior to admission, when he had noticed weakness of his legs and shortness of breath on exertion. This had increased and the patient had been confined to bed since that time. The patient gave no history of pain or discomfort after meals and neither nausea nor vomiting had occurred. There was no history of acute rheumatism. The patient denied having suffered from syphilis, but his blood had reacted to the Wassermann test.

On admission the patient had been well nourished, his colour had been good and the tongue moist and firm. The pulse had been rapid, regular and soft and the vessel walls had not been thickened. The cardiac sounds had been rapid and a systolic murmur had replaced the first sound at the aortic area. It had been heard best at the left border of the sternum in the third intercostal space. The first sound at the mitral area had been reduplicated. The breath sounds had been vesicular in type and the percussion note had been impaired at the right base posteriorly. Crepitations had been audible over large areas at both bases. The abdomen had been characterized by a fullness in the epigastrium. The liver had been greatly enlarged and its border had been palpable 7.5 centimetres (three inches) below the costal margin. It had been firm, no tenderness had been present and it had moved freely on respiration. No evidence of free fluid in the abdomen had been present. On examination of the nervous system it had been found that the pupils were equal and reacted to light and accommodation and the knee jerks were present. Some œdema of both ankles had been present.

On August 12, 1924, a blood culture had been made, but without result.

On August 27, 1924, a blood count had been made with the following results: Red cells 3,900,000 per cubic millimetre, hæmoglobin value 0.75%, colour index 0.9. It had also been noted on this day that the œdema of the legs was increasing, that the edge of the liver was nodular and hard and that a hard, painless mass was palpable in the epigastrium. Light diet had been given and a mixture containing iodide of potash, Fowler's solution, *liquor trinitrini* and tincture of digitalis had been taken every four hours. The patient had also taken 0.3 gramme of blue pill every fourth night.

On September 11, 1924, the abdomen had been more distended and the liver edge had reached the level of the umbilicus. Evidence of free fluid had been present in the abdomen. "Biereol" (an anti-syphilitic bismuth preparation) had been given in doses of 0.15 gramme every second day for ten doses.

On October 5, 1924, it had been noted that there was no diminution in the size of the liver. Œdema and ascites had increased and the patient's general condition was much worse.

Tuberculoma.

A widow, aged fifty years, an occupation manager of a confectionery business, had first been admitted to hospital on January 11, 1924, on account of pain in the back and lower limbs from which she had suffered for a period of two years. One week before admission she had complained of double vision and pain in the right side of the face. She had had no symptoms referable to the alimentary, circulatory, urinary or respiratory systems and no previous illnesses. The patient had suffered from pains in the legs and arms at intervals and these had been much worse since September 26, 1924. There was no history of headache or vomiting. The patient's husband had died of pleurisy twenty years previously and there was no family history of tuberculosis. It was pointed out that the patient was very intelligent and that her speech was very halting. Right hemi-anæsthesia and analgesia were present on the head and face and affected the bulbar conjunctiva as well as other parts. Attention was drawn to the fact that the notes made in March, 1924, indicated that the affected area had then been less extensive. Some right ptosis and strabismus were present. There was some hyperacusis of the right ear. The right side of the mouth sagged a little and the right masseter muscle was not as hard on contraction as the left. The posterior surface of the right thigh and the anterior surface of the right leg and of the left thigh appeared to be hyper-æsthetic. Bronchial breathing could be heard at the apex of the right lung and definite pleurisy was present at the left base. The reflexes were all present. The plantar reflex was flexor in type on the right side and doubtful on the left. No clonus was present. Some swelling was present in the right malar region. No reaction had been obtained either in the blood or cerebro-spinal fluid when the Wassermann test was applied. No tubercle bacilli had been found in the cerebro-spinal fluid or in the sputum.

Dr. A. W. Campbell had examined the patient and had reported as follows:

> The dominant neurological signs are complete paralysis of the fifth and sixth nerves and with this there is some swelling in the malar region. I think there is a growth (probably tuberculoma) in the sphenomaxillary fossa catching the above mentioned nerves as they cross, but I did not examine the discs for signs of intracranial pressure, namely papillœdema, which if present bilaterally or even unilaterally, would point to intra-cranial situation (Gasserian ganglion region).

Facial Erysipelas.

A male patient, aged thirty-four years, a cook, had been admitted to hospital on October 4, 1924. The patient had stated on admission that he had felt "off colour" on September 30, 1924. He had been attacked by pain in the ears and a general feeling of weakness and shivering. On October 2, 1924, the right side of the face had become swollen. He had been treated medically, but the condition had progressed. There was no history of injury to the face or the right ear or of discharge from the latter. There was a doubtful history of a small swelling in the left ear on September 27, 1924, which had burst and discharged. On admission the patient had been affected by erysipelas of the whole face. A definite raised edge had been present with the eruption. The right ear had been involved and was very tender to touch. It had been thought that there was furuncle present in the right ear. Blood stained discharge had been present from the left ear. The tongue had been heavily coated and moist. The fauces had been much injected and covered with mucus. Nothing abnormal had been found in the heart or lungs. The temperature had been 37.6° C. (99.8° F.), the pulse rate had been 120 and the respirations had numbered 24 in the minute. The urine had been acid and its specific gravity 1025. A faint cloud of albumin had been present. A furuncle in the right external auditory meatus had been incised and gauze inserted for a few hours. The patient had been given milk and milk foods.

On October 5, 1924, it had been noted that the temperature was very high and that the inflammation was spreading. Sixty cubic centimetres of anti-streptococcic serum had been injected intramuscularly. A 20% solution of "Argyrol" had been dropped into the eyes three times a day. The ears had been swabbed with peroxide of hydrogen and saline solution every two hours and the face had been dressed with a 15% solution of "Ichthyol" in glycerine twice a day and covered with a mask.

On October 6, 1924, the inflammation had extended and sixty cubic centimetres of anti-streptococcic serum had again been injected intra-muscularly.

On October 7, 1924, the high temperature had still been maintained and a third injection of the same dose of anti-streptococcic serum had been given.

On October 8, 1924, the temperature had become normal in the morning, but had risen again at night. The condition of the face had begun to subside, no pain was present and the general condition had improved.

On October 9, 1924, the day of demonstration, further improvement was noted, the temperature was normal and the pulse rate 72.

Endocarditis.

A male patient, aged thirteen years, a schoolboy, had been admitted to hospital on July 16, 1922, with a history of sore throat, shivering and pains in the joints of a few days' duration. The temperature had been 38.6° C. (101.4° F.). The right knee had been swollen and the heart had been apparently normal.

On May 25, 1922, a to-and-fro murmur had been detected at the aortic area, this had been conducted down the sternum and into the vessels of the neck.

On May 27, 1922, a mitral systolic murmur conducted into the axilla had been found.

On May 31, 1922, the patient's condition had been diagnosed as malignant endocarditis and the prognosis had been regarded as hopeless.

On June 6, 1922, blood culture had proved sterile and the patient's temperature had still been high.

On June 13, 1922, a pericardial friction sound had been heard.

On June 16, 1924, the to-and-from murmur had been audible at the lower end of the sternum only. The patient's condition had gradually improved and at the end of July, 1922, the heart had appeared to be normal and the patient was discharged.

Subsequently the patient had been well for two years and able to go to school. On one occasion he had had tonsilitis and was confined to bed for two days.

On July 30, 1924, the patient had been re-admitted to hospital with a diagnosis of acute appendicitis. On admission the temperature had been 38.6° C. (101.5° F.). The heart and lungs had appeared normal and the abdomen clear. The left tonsil had been enlarged and inflamed. A Widal test had been carried out, but no result obtained. The serum had not reacted to the Wassermann test. The urine had been normal and sterile on culture. No Klebs-Löffler bacilli had been found in the throat.

On August 4, 1924, a to-and-fro aortic murumr had been audible and the blood had been sterile on culture.

On August 14, 1924, diastolic murmurs at the base had conducted down the sternum, systolic and diastolic murmurs had been audible at the mitral area.

On September 30, 1924, the apex beat had been in the sixth intercostal space and 1.25 centimetres outside the nipple line. The aortic to-and-fro murmur had still been audible.

On October 8, 1924, the patient had been seen by Dr. Hamilton Marshall who had found only an aortic diastolic murmur and little, if any, enlargement of the heart. Dr. Marshall had recommended the early removal of the left and only remaining tonsil; he considered the prognosis to be good.

Chorea Following Two Attacks of Acute Rheumatism.

A male ptaient, aged sixteen years, had been admitted to hospital on October 8, 1924. The patient had stated that he had had an attack of pain and swelling in the right ankle and knee joints two years previously. He had been in bed on and off for seven weeks under treatment. The patient had felt quite well until three months before admission when he had been seized with severe cramping pain over the heart after he had been at work for two hours (he was apprenticed to a cabinet maker). He had experienced great difficulty in respiration on account of the pain. He had undergone medical treatment at home and later in hospital for one month. He had been discharged on September 30, 1924, and two days later had suffered from twitching of the right arm and dragging and twitching of the right leg. There was no history of sore throat or of fits. The patient had been injured in the head while a child.

It was pointed out that the patient was an intelligent, healthy-looking youth. He answered questions readily and well. Choreiform movements of the right arm and leg and of both sides of the face were present, the facial movements were more noticeable on the right side. The movements increased with excitement or when the patient was asked to fix the limb in space. The movements were purposeless and involuntary in character. Definite paresis of the right arm and leg were present. The right pupil was more dilated than the left and both pupils reacted to light and accommodation. The knee jerks on the right side were sustained and the plantar reflex was flexor in type. The tongue was clean. The fauces were injected and the crypts of the tonsils contained some exudate. The teeth were good. The pulse was regular and of small volume and its rate was 80 in the minute. The systolic blood pressure was 110 millimetres of mercury and the diastolic pressure 55 millimetres. The cardiac dulness was within normal limits and the sounds were regular. At the mitral area the first sound was partly replaced by a systolic murmur and the second sound was followed by a short diastolic murmur sometimes continuous with a presystolic murmur running up to the first sound. A soft diastolic murmur was audible in the third and fourth intercostal spaces on the left side near the sternum. No other abnormality could be detected in the chest. Treatment would be carried out by giving a full diet with extra milk and by the weekly intravenous injections of 0.3 gramme of "Salvarsan." The patient would be kept absolutely quiet.

Leucoderma.

A male patient, aged thirteen years, had been admitted to hospital on September 19, 1924, on account of a septic condition of the foot. The patient manifested a condition of leucoderma on the limbs and trunk. White areas devoid of pigment were surrounded by skin in which the pigment was increased. The mother stated that no sign of the skin condition had been present until after an attack of scarlet fever in England three years previously.

Vesical Calculus.

A male patient, aged eighteen years, a labourer, had been admitted to hospital on October 2, 1924, suffering from a urethral discharge of two months' duration. The discharge had been regarded as gonorrhœal in nature. The patient had denied coitus at any time. For four months he had had difficulty in micturition and the act was becoming increasingly painful. At the time of admission pain during micturition had been practically continuous and intense at conclusion of the act. The urine had come away "in fits and starts" and there had been sudden, painful stoppages; urine was passed every hour. The pain had not been increased by exertion or relieved by rest. It had been noted on admission that there was a thick white urethral discharge, but there was no inflammation of the urethra, that there was no abnormality in the testes or epididymes and that pressure on the perineum caused pain. On rectal examination the prostate had appeared normal, but definite tenderness had been present on palpation. Examination of the abdomen, heart and lungs had revealed no abnormality.

On October 3, 1924, a soft catheter had been passed without difficulty and the bladder capacity found to be fifteen cubic centimetres (half an ounce). The urine had contained numerous pus cells, but no tubercle bacilli had been found. No gonococci had been found in urethral smears.

On October 6, 1924, a sound had been passed into the bladder and a definite grating had been felt on withdrawal of the instrument. On X-ray examination a large irregular calculus about 6.25 centimetres (two and a half inches) in diameter had been found. Dr. R. J. Silverton had confirmed the X-ray report by cystoscopy and the stone would be removed by suprapubic operation.

Bronchiectasis.

A male patient, aged ten years, had been admitted to hospital on September 2, 1924. The patient had been in the Coast Hospital twice before and had then coughed up three hundred cubic centimetres (ten ounces) of pus on two occasions. The patient had had tracheotomy performed at the Royal Alexandra Hospital for Children six years previously whilst suffering from laryngeal diphtheria. He had not felt well since and suffered from recurring attacks of fever, pain in the right side of the chest and profuse sputum. He had got very short-winded on exertion. Nine days before admission the patient had had "a turn" with pain in the right side of the chest, blood stained sputum and anorexia. On admission it had been noted that the patient was an intelligent, old-fashioned boy. His lips were cyanosed, the colour of the cheeks was good, the tongue was clean, definite clubbing of the fingers and great toes was present and the limbs were very thin and wasted. It had also been noted that the child was pigeon chested, the right side was collapsed and moved less than the left. The percussion note over the right side of the chest was impaired, especially in the lower half. The note on the left side was hyperresonant. Tubular and bronchial breathing were present in the right lung and rhonchi and râles of medium and coarse type were present on the right side. The sounds in the left lung were normal. The sputum was profuse, purulent and very offensive. It was also pointed out that tubercle bacilli had not been found in the sputum and that the patient was losing weight. The radiologist had reported the presence of density at the right base and this was regarded as possibly due to thickened pleura with abscess formation. Dr. Hamilton Marshall had seen the patient and had advised operation with the production of artificial pneumothorax.

Acute Miliary Tuberculosis.

A male patient, aged twenty-nine years, a labourer, had been admitted to hospital on July 26, 1924, with a diagnosis of ascites. The patient had complained of abdominal pain, vomiting and loss of weight for the previous five weeks. Prior to this he had been quite healthy. He was a teetotaller. He had suffered from trench fever during four years' active service. He had not suffered from venereal disease. The onset of his illness had been characterized by sudden epigastric pain and vomiting. The pain had been of an intermittent nature, the attacks lasting as long as twenty-four hours. Vomiting had occurred daily and had afforded no relief to pain. The pain had had no relation to the ingestion of food. Three weeks before admission his abdomen had begun to swell. The vomitus had consisted of recently eaten food and no blood had been present. The stools had been normal. There had been no symptoms referable to circulatory, respiratory or nervous systems. The patient had been sallow, wasted and cachetic. The tongue had been coated, some pyorrhœa had been present. The abdomen had been distended and the liver normal. No free fluid had been found in the peritoneal cavity. The appearance of the patient had suggested that he was suffering from a severe form of typhoid fever. The leucocyte count had been four thousand cells per cubic millimetre.

The Widal test had been carried out on several occasions. On more than one occasion a definite tendency to agglutination had been present. A report had been obtained from the Bureau of Micro-Biology that the *Bacillus typhosus* had not been detected in either urine or fæces.

The patient had died suddenly on August 8, 1924, after a brief attack of acute abdominal pain.

A *post mortem* examination had been carried out and the specimens were demonstrated at the meeting. It had been found that the visceral and parietal peritoneum were studded with miliary nodules. The ileum had been perforated in three places. Typical tuberculous ulcers had been found in the ileum transversely round the bowel. In some places the ulcers had caused stricture. The spleen had been studded with nodules. The kidneys and liver had been normal. Miliary nodules had been found in both lungs, but no consolidation or cavitation were present. Caseous glands had been found in the mediastinum.

Tabes Dorsalis: Charcot Joints and Spondylitis.

A male patient, aged fifty-seven years, a clerk, had been admitted to hospital on September 8, 1924. He had given a history of a primary sore, syphilitic in nature, twenty years previously. He had been treated with medicine, but up to the time of admission had received no injections of any kind. Ten years previously his right knee had given way in the street and since that time he had walked with an iron in his boot. The knee had not been painful. Four months before admission he had begun to complain of shooting pains in the left calf and thigh and two weeks later had had an attack of vomiting and diarrhœa which lasted for four days. A week before admission he had complained of shortness of breath and blood stained fluid had been aspirated on two occasions from the right pleural cavity.

It was pointed out that definite scoliosis of the lumbar and lower dorsal regions of the spine was present. A Charcot type of knee joint was present on the right side with exostosis and destruction of the upper end of the tibia. Skiagrams were shown to demonstrate this condition. Skiagrams were also shown to demonstrate the osteo-arthritic changes in the left knee joint. The pupils reacted sluggishly to light and the knee jerks could not be elicited. Some paræsthesia of the skin of the feet was present, but otherwise sensation was normal. The blood serum had not reacted to the Wassermann test.

Hydronephrosis.

A female patient, aged sixty-six years, a cook, had been admitted to hospital on August 19, 1924, complaining of a tumour in the right side. Seven years previously the patient had noticed a swelling and had experienced an aching pain in the right side of the abdomen. The swelling had disappeared on the passage of a large quantity of urine after the application of hot fomentation. The patient had suffered from incontinence of urine since her last confinement seventeen years previously. Examination on admission had revealed the presence of a large mass in the right hypochondrium. It had been tense on palpation, dull on percussion, had moved with respiration and had not been tender on pressure. The urine had contained blood, pus and albumin.

On October 7, 1924, the tumour on the right side had become definitely diminished in size. A similar tumour had appeared on the left side, tense, semi-fluctuant, movable and not tender on pressure. The urine had contained pus cells and on culture had yielded a coliform bacillus. No calculus had been found on X-ray examination. The leucocytes had numbered 10,000 cells per cubic centimetre. The result of the urea concentration test had been 1.7%.

Enteric Fever.

A female patient, aged twenty-three years, had been admitted to hospital on September 2, 1924, with a history of three weeks' illness. A reaction had been obtained to the Widal test on the day after admission. The leucocytes had numbered 7,000 per cubic millimetre and the temperature had been over 39.7° C. (103.4° F.). On this day the patient had been given an intravenous injection of one cubic centimetre of typhoid, para-typhoid *A* and *B* vaccine standardized to 1,500 million organisms per cubic centimetre. The vaccine had been diluted with saline solution for the purposes of injection. A considerable reaction with a rigor had resulted and nine hours after the injection the temperature had been 40.1° C. (104.2° F.). On the following day the same dose of a similar vaccine had been given by intra-muscular injection. No rigor had resulted, but the temperature eight hours after the injection had been 40.5° C. (104.8° F.). Two days after the second injection the temperature had been 39.2° C. (102.6° F.) at 6 o'clock a.m., but normal at 2 p.m. and 6 p.m.. After this recovery had been steady and uneventful with scarcely any recurrence of the pyrexia. On September 18, 1924 (fourteen days after the last injection) the *Bacillus typhosus* had not been found on examination of the urine and fæces.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on October 3, 1924, DR. V. MCDOWELL, the Vice-President, in the chair.

Kidney Function in Health and Disease.

DR. J. V. DUHIG read a paper entitled "Kidney Function in Health and Disease" (see page 617).

DR. J. LOCKHART GIBSON congratulated Dr. Duhig on his paper. He was quite competent to criticise its biochemistry. Regarding prognosis in chronic nephritis, there was one condition which unfortunately enabled them to prognosticate with certainty, that was albuminuric retinitis. He had never himself known a patient live more than eighteen months after albuminuric retinitis with white exudation around the disc and retinitic patches in the neighbourhood of the macula with or without hæmorrhages, had supervened. He knew some observers had reported patients to live for a much longer time. It was particularly sad to have a young man or a young woman presenting at the consulting room, often looking very well and often without having had any medical advice, but complaining of failing vision. The finding of albuminuric retinitis supported by examination of the urine, forced the conclusion in such cases that death would occur within two years. He remembered only one patient who had survived as long as three years after slight retinitis had been found in the neighbourhood of one macula accompanied by slight albuminuria, but that patient had not lived a year after patches of white exudation appeared around the discs. He wished to insist that albuminuric retinitis was a neuro-retinitis with little or no swelling of the discs, unless of course cerebral symptoms were added and that papillœdema was quite a different condition with a different pathology, namely a result of increased intracranial tension and due to increased secretion of the chorioid plexus or gland.

He would ask Dr. Duhig whether it was possible to explain, except as he himself was accustomed to explain it, that whereas albuminuric retinitis associated with chronic nephritis was followed so certainly by death, yet in the albuminuric retinitis of pregnancy the prognosis was favourable, provided the uterus was emptied without delay. The appearances in the fundus were practically identical, the vision equally reduced, but prompt emptying of the uterus led to a clearing up of the fundus and restoration of perfect sight and perfect health.

He mentioned one patient whom he knew to be well after thirty years. However much it might be right to endeavour by diet and so forth to reduce toxæmia and restore disordered kidney function during pregnancy, the time for doing this had passed when albuminuric retinitis supervened.

He (Dr. Gibson) spoke in the strongest terms of the culpability of any delay in the emptying of the uterus, when albuminuric retinitis appeared. In a pregnant woman the appearance of failing vision should lead to an examination of each fundus and especially so if the urine contained albumin. He had within the last two years seen a case where examination of the eyes had been neglected in spite of diminished vision and failure to induce labour had resulted in permanently damaged fundi and vision with permanently damaged kidneys and health. She had been in a lying-in hospital with medical supervision, not in Queensland.

DR. S. F. MCDONALD said that he had listened to Dr. Duhig's paper with very great interest and had enjoyed the clarity with which he had put forward the results of many different workers in a concise scheme. The work of the bio-chemist had made much that was previously unintelligible, clear especially in regard to renal efficiency. He differed a little from Dr. Duhig as to the cause of nycturia in chronic nephritis. In such cases the urea concentration was low. The specific gravity was low and the kidneys' only way of responding to the stimulation of the increased head of nitrogenous waste in the blood was by pouring out a very dilute urine in large quantities. Even so this could not be accomplished during the working hours and so the kidney was unable to share in the general body rest of sleep and had to work on overtime. It was only by functional tests that they could say definitely whether nephritis or merely albuminuria was present and only by functional tests could they determine the degree of damage. They had in Queensland some hundred post-nephritic soldiers. In fifty of these no test could disclose renal damage, but many were chronic invalids especially from pain in the back. Of the remaining fifty about thirty were suffering from mild defects, whilst the remaining twenty were in all stages of breakdown. Even these with care and comfort were doing better than would be expected. The final clinical symptom in patients who had long shown signs of renal insufficiency, was albuminuric retinitis and usually the termination was less than six months distant.

DR. J. A. CAMERON expressed the opinion that the ocular symptoms during pregnancy might not be due to any renal lesion, but to pressure symptoms caused by swelling of the infundibulum.

DR. A. J. MACDONALD did not agree with Dr. Gibson that albuminuric retinitis was not associated with papillœdema. He was treating a patient so affected. He referred to Foster Moore's figures, namely that ninety patients with albuminuric retinitis had an average duration of life of three years, the maximum being seven years. The usual mode of daeth was by a hæmorrhage into the brain or some other region. Retinitis like the vomiting of pregnancy was due to a toxic condition of the fœtal membranes and there was no necessity to terminate pregnancy. The condition might not recur in future pregnancies.

DR. E. S. MEYERS inquired as to the frequency of ocular symptoms in persons suffering from the albuminuria of pregnancy.

DR. A. C. F. HALFORD gave the clinical history of a patient who had been in the seventh month of pregnancy. She had passed urine containing "solid albumin," had suffered from albuminuric retinitis and had had a blood pressure over of 300 millimetres of mercury. The uterus had been emptied, but no improvement of symptoms had occurred. The albumin had disappeared from the urine after six months and there had been a slight reduction in blood pressure. The difference between the neuritis of a primary kidney condition and that of pregnancy lay in the fact that the cause could be removed in the latter, but not in the former as it was unknown. The nephritis of young adults was the result of mild or unrecognized scarlet fever.

DR. R. MARSHALL ALLAN, M.C., said that in Dublin all patients with albuminuria of pregnancy were strictly dieted. If improvement took place, the pregnancy was allowed to continue. But in severe toxic conditions accompanied by definite eye symptoms which did not react to treatment or which steadily grew worse, the pregnancy was terminated.

DR. J. V. DUHIG in reply thanked the members for the reception accorded to his paper and said that the question of pregnancy was still *sub judice*. The toxin might be primary or secondary. According to the opinions of several authors recorded in *The Journal of Obstetrics and Gynæcology of the British Empire*, Volume XXXI., No. 1, albuminuria of pregnancy might be due to a derangement of the liver function.

Pathological and Other Specimens.

DR. J. V. DUHIG exhibited a number of specimens. These included the spleen of a girl, aged seventeen years, who had suffered from splenomegaly. A sister of the patient had died at the age of sixteen years, two years previously from an illness with similar symptoms. The patient had been a tall, strong girl. Her skin had been faintly brown in colour and somewhat "muddy" in appearance. The spleen had been greatly enlarged and had extended beyond the level of the umbilicus as far downwards as the pelvis on the left side. It had been firm and rounded. The liver had been palpable below the costal margin. The blood count had revealed no abnormality beyond a slight diminution of the erythrocytes and a slight increase in the number of leucocytes. The Wassermann test had been applied to the blood serum, but no reaction had been obtained. A distinct "indirect" reaction had been obtained to the Van den Bergh's test (non-obstructive jaundice). It had been found that hæmolysis took place readily when the blood was diluted with 0.475% sodium chloride; a trace of hæmolysis occurred with 0.5% sodium chloride. Brill and Mandelbaum's conjunctival sign had not been elicited. The diagnosis of Gaucher's disease had been suggested on account of the familial character of the affection and some of the other findings. On the other hand the rarity of this disease had induced the surgeons to adopt a cautious attitude in regard to the diagnosis. The spleen had been removed. It had weighed about three kilograms. Much blood had escaped from the organ, with the result that it had shrunk considerably. Originally it had had a dull red colour; after removal it had assumed a dusky red. Great increase of reticulation tissue with atrophy of the pulp and almost complete ablation of the Malpighian bodies were noted on section of the organ. These were appearances resembling the changes seen in the spleen in Banti's disease. Accordingly the diagnosis of Banti's disase was made. The patient had died.

The second specimen was a piece of intestine affected with multiple tuberculous ulceration. He also exhibited a very large carcinomatous tumour of the stomach, a renal sarcoma from a child and several kidneys to illustrate the changes met with in sub-acute and chronic nephritis referred to in his paper on "Kidney Function in Health and Disease" (see page 617). He also showed a "myelin" kidney the result of thrombosis caused by massive staphylococcus infection, some specimens of *Trichuris trichuria* from a boy with a triple infestation, the other worms recovered being *Necator americanus* and *Anchylostomum duodenale* and *Strongyloides stercoralis*, a massive infestation by *Ascaris lumbricoides* in a boy aged ten years and lastly hookworms from a patient whose infestation had been 100% *Anchylostomum*. No *Necator* worms had been recovered from this patient, a resident of the northern part of Queensland.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

OWEN, HYAM MAURICE, M.B., Ch.M., 1924 (Univ. Sydney), 58, Cambridge Street, Stanmore.

FINLAY, CUTHBERT CLIVE, M.B., 1923 (Univ. Sydney), "St. Berner," Kemmis Street, Randwick.

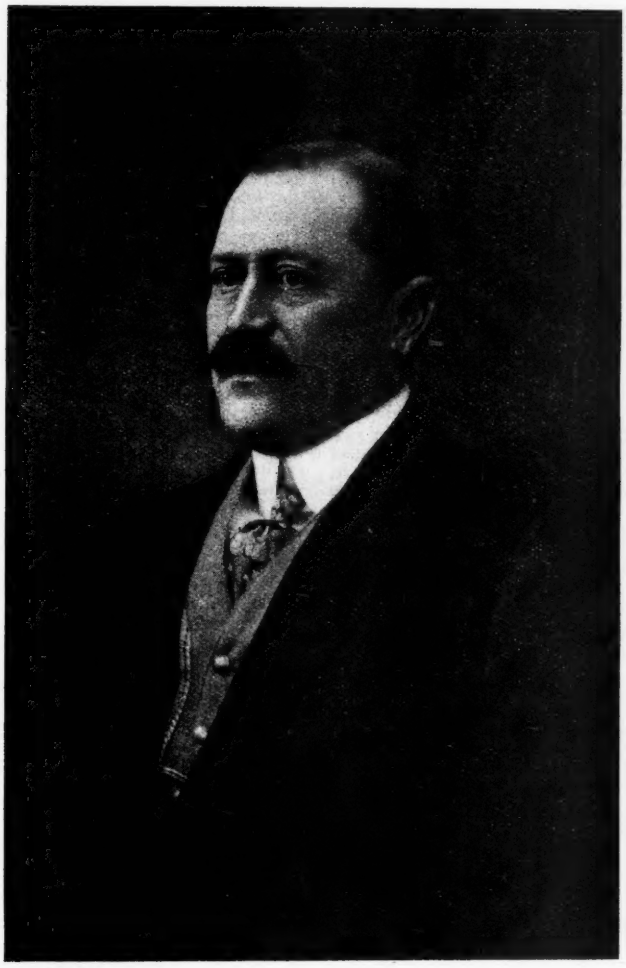
Obituary.

ROWLAND EDWARD HARROLD.

IN our issue of November 29, 1924, we announced the death of Rowland Edward Harrold. For a considerable time his friends and acquaintances felt anxious concerning the state of his health, but few anticipated that his illness would have an early fatal termination. He died on November 6, 1924, at his home in East Terrace, Adelaide.

Rowland Edward Harrold was born in Adelaide in 1855 and was consequently sixty-nine years of age. His education was conducted first at a private school owned by Mr.

John L. Young and later at the Glenelg Grammar School. He then matriculated and entered the University of Adelaide where he began his medical studies. He then visited Scotland and became a student at the Edinburgh University. In the year 1890 he took the degrees of Bachelor of Medicine and Master of Surgery. Soon after graduating he went to London and turned his attention to the study of laryngology. He accepted the position of clinical assistant to the late Morell Mackenzie at the Golden Square Throat Hospital. He served for one year during which time he went into residence at the hospital. In 1892 he returned to Australia and started practice early in the following year in Adelaide. During the ensuing eight years he built up a valuable practice and established for himself a reputation of being a very competent and kindly practitioner. He was very popular and did not spare himself any pains in his endeavour to serve his patients well. In 1910 he again visited England for the purpose of continuing his studies of dermatology. He worked under J. H. Sequeira and other dermatologists and rendered himself especially expert in this specialty. When he returned to Adelaide he relinquished general practice for diseases of the skin and very soon became recognized as an authority. He was appointed Honorary Dermatologist to the Adelaide Hospital. His reputation as a dermatologist was not limited to his own city; his colleagues in other States were well acquainted with him and his work and held both in high regard. At at earlier date he served as an honorary assistant physician in the Adelaide Hospital.



Rowland Edward Harrold has published many articles on subjects connected with his specialty, including *xanthoma diabeticorum* and *epidermolysis bullosa*. He took part in the discussions at the Australasian Medical Congress, Brisbane, 1920. He was selected as President of the Section of Dermatology of the Australasian Medical Congress (British Medical Association), Melbourne, 1923, and delivered a skilful and highly suggestive address before the section. He was a member of the British Medical Association and took a keen interest in its activities.

We have already referred to Rowland Harrold's popularity among his patients. With his colleagues he was a general favourite. His kindly and artistic disposition secured for him a large circle of friends both inside and outside the profession. He was an expert horticulturist and his love for flowers was an indication of his humane and benevolent character.

The sympathy of the medical profession is extended to Mrs. Harrold and to his daughter.

University Intelligence.

UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held at the University on December 1, 1924. The degree of master of surgery (Ch.M.) was conferred *in absentia* upon Dr. G. C. Taylor.

The Honourable Sir W. P. Cullen was re-elected Chancellor and Dr. C. Purser, Vice-Chancellor.

An invitation was received from the British Medical Association, Hong Kong, for the University to be represented at a Conference at Hong Kong University, in January, 1925.

A letter was received from the Honorary Secretary of the Australasian National Research Council relative to the provision of funds for the establishment of a Chair of Anthropology at the University of Sydney.

A donation of books to the Fisher Library was received from Mr. F. Leverrier on behalf of Captain Charvin. The gift was accepted with grateful thanks.

Dr. W. C. Mansfield was appointed to the Lectureship in Diseases of the Ear, Nose and Throat and Dr. R. L. Davies to the Lectureship in Gynæcology.

Dr. R. H. Todd was re-appointed Lecturer in Medical Ethics.

A letter was received from the Under Secretary, Department of Public Health, stating that the Government had decided to grant a subsidy of £1 for £1 at the discretion of the Minister to a maximum of £5,000 towards cancer research.

The following representatives of the Senate were appointed:

(*a*) University Sports Union Committee: Dr. G. H. Abbott and Professor A. E. Mills.

(*b*) Women's College Council: Mr. Justice Street and Dr. Abbott.

The following were appointed examiners for the Final Medical Examinations:

Medicine: Professor A. E. Mills, Dr. Cecil Purser, Dr. S. A. Smith.

Surgery: Professor F. P. Sandes, Dr. R. B. Wade, Dr. H. C. Stacy.

Obstetrics: Dr. J. C. Windeyer, Dr. S. H. MacCulloch.

Gynæcology: Dr. R. L. Davies, Dr. George Armstrong.

Clinical Medicine: Dr. C. B. Blackburn, Dr. H. J. Ritchie, Dr. J. M. Gill, Dr. Sinclair Gillies, Dr. J. I. C. Cosh, Dr. A. W. Holmes à Court.

Clinical Surgery: Dr. G. H. Abbott, Dr. Gordon Craig, Dr. A. Aspinall, Dr. C. E. Corlette, Dr. H. J. Dansey, Dr. S. J. McKelvey.

The Alvarenga Prize.

THE College of Physicians of Philadelphia annouces that the next award of the Alvarenga Prize will be made on July 14, 1925, provided that an essay deemed by the Committee of Award to be worthy of the Prize shall have been offered. The value of the Prize is about three hundred dollars. The essays may be upon any subject in medicine. They should represent an addition to the knowledge and understanding of the subject based upon either original or literary research. They must not have been published. They must be type-written and in English acceptable for publication without the necessity for editing by the Committee. Illustrations should be appropriately and correctly annotated with the next. Each essay must be sent without signature, but must be plainly marked with a motto and be accompanied by a sealed envelope having on its outside the motto of the paper and within the name and address of the author. The essay must be received by the Secretary of the College of Physicians of Philadelphia, 19, South Twenty-Second Street, Philadelphia, Pennsylvania, United States of America, on or before May 1, 1925.

We have already announced that the Alvarenga Prize for 1924 was awarded to Dr. Gordon Cameron, Melbourne, for an essay entitled "Pancreatic Anomalies: Their Morphology, Pathology and Clinical History."

Naval and Military.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Nos. 79, 82 and 84, of October 23, November 6 and 13, 1924:

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Promotion.—SURGEON LIEUTENANT (ACTING SURGEON LIEUTENANT-COMMANDER) DAVID SHIELDS PRENTICE, M.B., M.R.C.P., is promoted to the rank of Surgeon Lieutenant-Commander, dated 1st July, 1924.

AUSTRALIAN MILITARY FORCES.

First Military District.

Australian Army Medical Corps.

LIEUTENANT-COLONEL F. C. WOOSTER, D.S.O., is restored to the authorized establishment of Lieutenant-Colonels, 1st July, 1924.

Reserve of Officers.

CAPTAIN A. H. CROWLEY, is transferred from the Reserve of Officers, 3rd Military District, 10th October, 1924.

Second Military District.

Australian Army Medical Corps.

MAJOR F. L. BIGNELL, D.S.O., is restored to the authorized establishment of Majors, 1st July, 1924; MAJOR C. L. S. MACINTOSH, D.S.O., is transferred to the Unattached List, 1st October, 1924.

To be Lieutenant-Colonels, supernumerary to the establishment of Lieutenant-Colonels, with pay allowance of Major.—MAJOR W. L. KIRKWOOD, O.B.E., MAJORS (HONORARY LIEUTENANT-COLONELS) C. E. WASSELL, D.S.O., and H. V. P. CONRICK, D.S.O., MAJORS R. W. W. WALSH, D.S.O., A. L. BUCHANAN, B. C. KENNEDY and W. E. KAY, D.S.O., 1st August, 1924.

To be Majors.—CAPTAINS (HONORARY MAJORS) F. McINTYRE, M.C., W. EVANS, M.C., R. B. NORTH and E. H. RUTLEDGE, and CAPTAINS N. E. B. KIRKWOOD, M.C., T. F. BROWN, D.S.O., V.D., A. M. DAVIDSON, O.B.E., C. F. ROBINSON, M.C., A. J. COLLINS, D.S.O., M.C., K. S. PARKER, M.C., and A. J. MOLLISON, 1st August, 1924. MAJORS F. McINTYRE, M.C., E. H. RUTLEDGE, N. E. B. KIRKWOOD, M.C., T. F. BROWN, D.S.O., V.D., A. M. DAVIDSON, O.B.E., C. F. ROBINSON, M.C., A. J. COLLINS, D.S.O., M.C., K. S. PARKER, M.C., and A. J. MOLLISON to be supernumerary to the establishment of Majors, with pay and allowances of Captain, 1st August, 1924.

To be Captains (provisionally).—HAROLD McLEAN, HOLLINGWORTH, M.C., and GERALD ARCHBOLD, 1st November, 1924; LIEUTENANT-COLONEL W. E. KAY, D.S.O., is transferred to the Unattached List, 21st August, 1924; MAJOR A. J. COLLINS, D.S.O., M.C., is transferred to the Reserve of Officers, 1st October, 1924.

Reserve of Officers.

The resignation of CAPTAIN A. J. CORFE of his commission is accepted, 4th October, 1924.

MAJORS A. GOLDSTEIN and C. H. SHEARMAN are transferred from the Reserve of Officers, 5th Military District, 20th October, 1924; MAJOR F. W. HALL is placed on the Retired List, with the honorary rank of Lieutenant-Colonel, and with permission to wear the prescribed uniform, 1st October, 1924; CAPTAIN G. A. BLUMER, V.D., is placed on the Retired List, with the honorary rank of Major, and with permission to wear the prescribed uniform, 1st October, 1924.

AWARD OF THE COLONIAL AUXILIARY FORCES' OFFICERS' DECORATION.

Unattached List.

LIEUTENANT-COLONEL H. K. DENHAM, D.S.O.

Correspondence.

THE PATIENT'S PRIVILEGE.

SIR: Mr. Shelton's valuable paper on "Criminal Abortion and the Medical Profession" is avowedly written from the standpoint of the law of Victoria. It would be interesting to know, however, what degree of uniformity exists in the law of the other States, particularly on the question of the privilege of the patient.

In Victoria, as Mr. Shelton informs us, in a "civil suit, action or proceeding" a medical practitioner must refuse to disclose information "acquired in attending the patient," whether (I take it) that information be acquired from the patient's statements or the doctor's examination or treatment, unless the patient agree to such disclosure. In criminal cases, Victorian law confers no such privilege.

In New South Wales, apparently, the patient is not given this privilege in civil cases. Some of your readers (perhaps, like myself, after asking to be excused on the score of professional obligations) have been instructed by the presiding judge to disclose in civil court information acquired in attending a patient. I presume this would not happen in Victoria. In the other States, does the patient possess the privilege granted in Victoria?

Yours, etc.,

R. C. EDWARDS.

Dulwich Hill, New South Wales,
November 8, 1924.

[The privilege to which Mr. Shelton refers, is extended to the patient in Victoria and New Zealand. In all other parts of the British Empire a medical witness may be required to disclose information in court in civil as well as in criminal cases. Judges, however, usually will not press the witness, if the latter asks whether he is compelled to divulge professional secrets.—EDITOR.]

VENOMOUS SEA-SNAKES.

SIR: Dr. Kevin Byrne's letter in your issue of November 15, 1924 (page 539), containing a reference to sea-snakes, prompts one to state that all the true sea-snakes of the sub-family *Hydrophinæ* are highly venomous. They belong to the section *Proteroglypha*, the different members having the front teeth of the upper jaw grooved to conduct the venom from the poison gland to the wound inflicted. The bite, however, displays many punctures, as with the harmless land-snakes, and not the typical two-puncture mark. In *Platurus*, however, the poison fang is short and not followed by a series of simple teeth as in *Hydrophis*, but a very small single tooth which is frequently lost, is im-

planted at some distance behind the poison-fang. Death following the bite is not common, owing to the small gape and the difficulty these snakes have in grasping large objects. Most of the fatalities have occurred owing to fishermen mistaking them for eels and being bitten on the fingers when attempting to remove them from the hook. Captain Meetham records the case of a native fireman fishing in Indian waters, who hooked one and died in very great agony a few hours after being bitten in removing it. Russell and Canter have proved by direct observation that tortoises, other snakes and fish died from the bite in less than an hour and a man succumbed in four hours. Their deadly nature has frequently been proved. The reason that comparatively few accidents occur is because they are extremely shy and swim away on the slightest alarm, but when surprised in the submarine cavities which form their natural retreats, they will like any poisonous terrestrial snake dart at a pole. When out of the water, they attempt to bite every object near them, even turning round to wound their own bodies (Canter). The eye is small with a round pupil which is so much contracted by the light when the snake is taken out of the water, that the animal is blinded and is unable to hit any object it wants to strike (Günther). Lucas and Le Souëf relate that Tongan and other Pacific Islander natives, including children, play with these snakes when bathing, coiling them round their arms and necks as armlets and necklets, quite ignorant of or oblivious to their deadly character. In some instances these snakes have been identified as the highly venomous *Platurus colubrinus*. The food of sea-snakes consists of small fish, often well provided with spines. As all these animals are killed by the poison of the snake before they are swallowed and as their muscles are perfectly relaxed, their armature is harmless to the snake which commences to swallow its prey from the head and depresses the spines as deglutition proceeds (Günther).

The true sea-snakes are found through the Indian and Pacific Oceans, over a dozen species being found in Australian waters, chiefly in tropical areas. All the Australian species are marine, but one species of *Distira* inhabits the fresh water of Lake Taal, in Luzon, Philippine Islands. The species may ascend the streams far beyond the estuaries, but not beyond tidal influence into fresh water. The largest Australian species is *Distira grandis*, attaining a length of seven feet eight inches. *Distira stokesii* (Stokes's sea-snake) sometimes exceeds five feet and has been taken at Middle Harbour, Port Jackson. The commonest species is the yellow-bellied sea-snake, *Hydrus* (*Pelamydrus*) *platurus*, known also as *Pelamis bicolor*. Possibly Dr. Byrne's green and red sea-snake is referable to this species. Mr. J. R. Kinghorn, of the Australian Museum, has kindly informed me that the colour of this species varies from black to olive green and brown above and from lemon yellow to varying shades of brown below. Some of the browns may be so light as to appear reddish under water. Forty-one inches is a record length for this species and anecdotes of their attaining a length of ten feet are pure fiction. Incidentally the largest sea-snake of any species verified scientifically was twelve feet in length. The usual length of the yellow-bellied species is three feet. This is the commonest species in eastern Australian waters and is not infrequently washed on Bondi and Manly beaches by storms. It is quite helpless on land and cannot wriggle back into the water. It soon dies out of water.

Platurus colubrinus and *Platurus laticaudatus* are the ringed sea-snakes. They are well at home on the land also and may be seen on rocks or swampy ground far from the ocean. The former species is sometimes washed ashore on the beaches about Sydney.

The true fresh water snakes belong to the sub-family *Hemalospinæ* of the section *Opisthoglypha*. Some of the hinder fangs are grooved to conduct the poison from the venom-sac to the wound. The bite inflicted would show on each side a series of small punctures, with a larger one posteriorly. They are all venomous, but none dangerous. They are aquatic, but not solely so and may appear on land. Their main habitat is fresh water, but some even enter the sea. Some have a prehensile tail to attach themselves to weeds *et cetera* as they await thir food, consisting of fish, crustacea *et cetera*. As in the true sea-snakes and crocodiles the nostrils are on top of the snout, to enable them to breathe with their bodies practically entirely submerged. Waite gives four Australian species, all from Queensland and Northern Australia. These are *Hypsirhina macleayi*, *Cerberus australis* (the Australian bockadam), *Myron richardsoni* and *Fordonia leucebalia* (the white-bellied water snake), with alternate black and white bands above and white beneath.

Finally we have another fresh-water snake, belonging to the sub-family *Natricinæ* of the colubrine snakes and the section *Aglypha*, in which the teeth are all solid and the species consequently harmless. This species is *Tropidenotus picturatus*. It occurs in great abundance all over the north of Australia and down the eastern coast as far as the Clarence River. It is a close ally of the ringed snake, *Tropidenotus natrix*, of Europe, and is a favourite article of diet of the Australian aboriginals, especially about Port Darwin, Port Essington *et cetera*. They are most in evidence about dusk when the natives capture them plentifully. The species is not completely aquatic, but lives also considerably on the land.

In conclusion mention may be made of the fact that the highly venomous black snake of Australia (*Pseudechis porphyriacus*) is an expert swimmer and is often seen in the water.

Yours, etc.,

JOHN MACPHERSON.

Wyoming, 175, Macquarie Street,
Sydney, November 20, 1924.

KÖHLER'S DISEASE.

SIR: It has not been my fortune to meet with a case of this type. On reading the case reported in THE MEDICAL JOURNAL OF AUSTRALIA of November 22, 1924, it struck me that a quicker and more certain result would be obtained by complete exenteration of the bony substance of the navicular bone. Keep the walls of periosteum apart by salt packs and in a few weeks complete reformation should appear. In a case of tubercular disease of the tarsus I have obtained a good result after thus exenterating every tarsal bone and in osteo-myelitis of the tibia, I have had a patient walking without support four months after complete exenteration of the diaphysis.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street, Melbourne
(Undated).

ACUTE AND CHRONIC DELIRIUM.

SIR: In Professor Berry's interesting article on "Some Clinical Example of Physical and Mental Testing" in THE MEDICAL JOURNAL OF AUSTRALIA, of November 29, 1924, he mentions as examples of "ill-health of the neurones" delirium in acute fever, acute mania after anæsthetics, *delirium tremens et cetera*, all more or less acute deliriums, that is poisonings of the brain cells (or so-called insanities, if at all long continued). He does not mention chronic delirium.

As acute poisoning causes acute delirium (for example typhoid), so chronic poisoning causes chronic delirium. In my opinion many so-called insanities as well as neuroses are chronic deliriums and the search for early removal of the cause would cure the condition, whether the brain be super-normal, normal or sub-normal in its make-up and capacities. Any brain, as any heart, may get (shall I say?) "cloudy swelling."

Under-neuroned brains, unpoisoned, may be adequate to life; super-normal or normal brains, poisoned, may be inadequate to life; in the former as well as in the latter who have lived to their twenties without mental illness, one should certainly look for the new cause disturbing the previous balance.

In my experience dead teeth are a fruitful source of poisoning of varied character and very frequently attacking the nervous system, causing neuroses and even psychoses or threatened "nervous break-downs." Many complain of sleeplessness, fatigue, tremors, fears, fears of going mad, irritability, dislike of relatives and of being upset by trifles which they know to be trifles; and such symptoms are relieved by extractions and eliminative medicines *et*

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cetera. Other septic foci in nose, throat, or ears may be responsible, but these have not come within my personal experience.

To my mind Case II. is an instance of chronic delirium; no family history or personal history of health, teeth *et cetera* nor even of mental condition before this illness is given; but she is rather inconclusively placed within the normal limits of morphological variation, though on the low side and, as she lived to her twenties apparently adequate to her life conditions, I feel sure something happened to upset her balance and it may have been something poisonous and not yet destructive. If she were my patient I would extract every tooth that had ever been carious and if apparently edenulous, would "X-ray" her jaws to determine whether any tooth fragments or abscess were still present and if these examinations were negative, would search for other chronic foci of poisoning.

It is most important for the general practitioner and the specialist to realize that brain poisoning may be acute or insidious in its onset and may be due to chronic as well as acute poisons and that "nervy" symptoms should be treated seriously as early indications of trouble and that mental or insane symptoms can yet clear up if a cause can be found and removed. I even go so far as to think that the increasing prevalence of insanity is due to chronic and neglected poisoning from dead teeth.

Yours, etc.,

MARY C. DEGARIS, M.D., B.S. (Melb.).

"Gable House,"
268, Latrobe Terrace, Geelong,
November, 30, 1924.

Books Received.

MANUAL OF SURGERY FOR STUDENTS AND PRACTITIONERS, by Albert Carless, C.B.E., M.B., M.S. (London), F.R.C.S.; assisted by Cecil P. G. Wakeley, F.R.C.S., Eleventh Edition; 1924. London: Baillière, Tindall and Cox. Demy 8vo., pp. xii. + 1600, with illustrations and X-ray supplement. Price: 30s. net.

REPORT ON MINERS' "BEAT KNEE," "BEAT HAND" AND "BEAT ELBOW," by Professor E. L. Collis, M.D., M.R.C.P., and T. L. Llewellyn, M.D.; Medical Research Council (Privy Council), Special Report Series, No. 89; 1924. London: Published by His Majesty's Stationery Office.

SOCIAL PATHOLOGY, issued by the United States Public Health Service. Volume I., Nos. 1—4.

TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, Edited by Walter G. Elmer, M.D.; Third Series; Volume XLV.; 1923; Philadelphia. Royal 8vo., pp. lvii. + 856.

THE BRITISH JOURNAL OF SURGERY: GENERAL INDEX TO VOLUMES I.—X., from July, 1913, to April, 1923, Numbers 1—40. Compiled by Archibald L. Clarke; 1923. Bristol: John Wright and Sons, Limited. Crown 4to., pp. 170.

THE ERRORS OF ACCOMMODATION AND REFRACTION OF THE EYE AND THEIR TREATMENT: A HANDBOOK FOR STUDENTS, by Ernest Clarke, M.D., F.R.C.S., Fifth Edition; 1924. London: Baillière, Tindall and Cox. Crown 4to., pp. iv. + 251, with illustrations. Price: 8s.6d. net.

THE EXTRA PHARMACOPŒIA OF MARTINDALE AND WESTCOTT, Revised by W. Harrison Martindale, Ph.D., F.G.S., and W. Wynn Westcott, M.B. (London), D.P.H. Volume I., Eighteenth Edition; 1924. London: H. K. Lewis and Company, Limited. Foolscap 8vo., pp. 1163. Price: 27s. 6d. net.

THE NERVOUS PATIENT, by Millais Culpin, M.D. (London), F.R.C.S. (England), with a Chapter on "The Major Psychoses," by Dr. Stanford Read and on "Eye Symptoms," by Mr. W. S. Inman; 1924. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. viii. + 305. Price: 10s.6d. net.

ULTRA-VIOLET RAYS IN THE TREATMENT AND CURE OF DISEASE, by Percy Hall, M.R.C.S. (England), L.R.C.P. (London), with Introductions by Sir Henry Gauvain, M.A., M.D., M.C. (Cambridge), and Leonard E. Hill, M.B. (London), F.R.S.; 1924. Lonndon: William Heinemann (Medical Books) Limited. Royal 8vo., pp. 110, with illustrations. Price: 7s. 6d.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi..

UNIVERSITY OF ADELAIDE: Marks Lectureship in Applied Physiology and Sheridan Fellowship.

BROKEN HILL AND DISTRICT HOSPITAL, NEW SOUTH WALES: Senior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

DEC. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee. Organization and Science Committee.
DEC. 24.—Victorian Branch, B.M.A.: Council.
DEC. 26.—Queensland Branch, B.M.A.: Council.
1925.
JAN. 6.—New South Wales Branch, B.M.A.: Council (Quarterly).
JAN. 8.—Victorian Branch, B.M.A.: Council.
JAN. 10.—Queensland Branch, B.M.A.: Council.
JAN. 10.—South Australian Branch, B.M.A.: Council.
JAN. 13.—New South Wales Branch, B.M.A.: Ethics Committee.
JAN. 14.—Tasmanian Branch, B.M.A.: Branch.
JAN. 17.—Northern Suburbs Medical Association, New South Wales.
JAN. 20.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 22.—Victorian Branch, B.M.A.: Council.
JAN. 24.—Queensland Branch, B.M.A.: Council.
JAN. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
JAN. 28.—Victorian Branch, B.M.A.: Council.
FEB. 3.—Tasmanian Branch, B.M.A.: Council.
FEB. 6.—Queensland Branch, B.M.A.: Branch.
FEB. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 12.—Victorian Branch, B.M.A.: Council.
FEB. 12.—South Australian Branch, B.M.A.: Council.

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